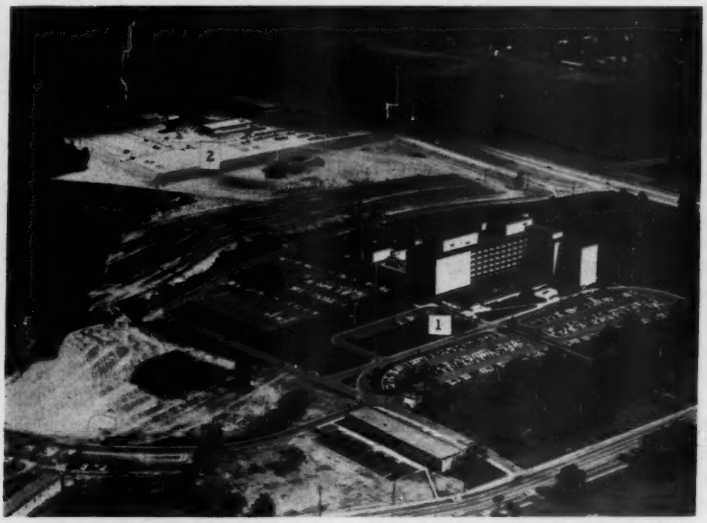
THE AMERICAN SURGEON

Vol. 27, No. 3 March 1961



1. University of Mississippi Medical Center. 2. Veterans Administration Center.

FOREWORD

The Department of Surgery of the University of Mississippi Medical Center is pleased to present these issues of the journal. Established in 1955, the department set for itself objectives which included effective undergraduate and postgraduate teaching, the care of patients in a superior manner, the establishment of a sound residency system and the development of a spirit of inquiry which would permeate the entire effort and serve to provide direction, purpose and intellectual stimulation for all. The success of the program has been gratifying. Through the energetic efforts of the full time and attending staff, it has been possible to provide effective departmental activity at all levels. The result has been an orientation of students, internes, residents and staff in the direction of critical analysis not only of physiologic and diagnostic problems, but also of technical operative procedures. This attitude has provided a challenging environment in which all might work.

The articles appearing herein serve to indicate something of the nature and the range of the clinical and investigative interests of various members of the department of surgery.

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SUGGESTIONS FOR AVOIDING MALPRACTICE SUITS*

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The rapid increase in malpractice suits, both in number and size of monetary awards, makes it highly desirable that surgical residents be given training in avoiding malpractice suits. In this article a few of the many aspects of this problem will be discussed.

Malpractice actions are usually governed by state laws which vary considerably from state to state. Consequently an article such as this must be written in generalities.

WHO IS SUED?

According to a review by the American Medical Association legal staff,[2] no one class of physicians is immune from such a suit. One out of every seven doctors in the United States has had a malpractice claim or suit brought against him. Sixty per cent of these have been decided against the physician.

California, home of the noted malpractice lawyer, Melvin Belli,[4] has the highest rate. Here one out of every four doctors has had a claim or suit. The lowest rate is in South Carolina, where about one in 33 physicians has been involved. The other states fall in between, with New York second and Ohio third.

Female physicians are sued less frequently than male;[3] but of the persons starting the suits, 70 per cent are women.

The physician may expect his first malpractice claim after about 10 years in practice.

Approximately 50 per cent of the doctors sued are specialists, with the highest percentages being general surgeons, next the obstetrics-gynecology group, and third the orthopedists.

Internes and residents are frequently included in suits in teaching hospitals, along with the attending physician. It would be wise, before acceptance of interneship or residency, for the individual to determine whether the hospital provides insurance against malpractice, as many hospitals do.

DUTY TO PATIENT

The physician has no duty to a patient unless he agrees to treat him. For example, if he is the only available surgeon within 100 miles and he is called to stop a severe hemorrhage from a traumatic wound, he may be under a moral obligation to respond to the call, but not a legal one. If he refuses and the individual dies, he is not liable to the patient's survivors for his death.

If the surgeon assumes the duty to care for a patient, he is not relieved of this duty until the patient either is well, discharges him as his surgeon (and one should have this in writing), or is turned over to another physician.

For example, a surgeon traveling on vacation stops at an auto accident and applies a splint to the leg of an accident victim with a closed fracture of the tibia and fibula. He waits until the ambulance arrives, sees that the patient is on a stretcher in the ambulance, and then continues on his vacation. On the way to the hospital the ambulance driver removes the splint from the leg. The fracture becomes an open one and there is severe soft tissue damage from the motion at the fracture site. It later becomes infected, and the leg has to be amputated.

In a case similar to this, it was held that the surgeon abandoned the patient and was liable for the poor result, since he turned him over to the ambulance driver instead of accompanying the patient to the hospital where he could have been released to another doctor. Some states have laws relieving the physician of liability in such situations. Again, there is no legal duty to stop and give first aid.

One must not overlook the fact that a physician's duty to the patient may be assumed in an indirect manner, such as being employed

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NOTE: The opinions expressed are those of the author and not those of the Veterans Administration.

as a plant physician, being designated for workmen's compensation cases, or being employed by some form of a group plan. The physician cannot refuse to treat such cases and must make a satisfactory disposition of the case.

PHYSICIAN-PATIENT RELATIONSHIP

We frequently hear that, with the advance of science, the "art of medicine" is being lost. This may be true in part, but only in part, because a month does not pass that one does not hear of a case which, because of complications, might form the basis for a large judgement in a malpractice suit. However, the patient still has utter confidence in his physician, despite a poor result from unfortunate treatment, and no legal action is even considered.

A new physician seeing such a patient should follow the golden rule. He should never criticize another physician in the presence of the patient. If he disenchants the patient with the first doctor, that physician may be sued. Twenty-five per cent of the malpractice suits are initiated because of derogatory comments made by physicians not involved in the case, according to a survey by the American Medical Association.[2]

Also, a physician may be sure that if he practices surgery for long, no matter how good he is, he will be in the other doctor's shoes not once, but many times over. There is no surgeon living who has had extensive surgical experience who has not many times obtained results which could have resulted in a malpractice suit against him.

The "art of medicine" includes the ability to have the patient accept a poor result, or, despite losing an elective operative case, to have the family's gratitude for all the effort. It comes from kindness and consideration for the patient, and from taking enough time to explain in an honest fashion the indications, the expected results, and the risks. By all means, a physician should never guarantee anything.

CONSENT FOR OPERATION

The mere fact that a patient signs a consent slip for operation does not give the surgeon immunity. The patient must understand what he signs. A woman who signs for an appendectomy and "any other indicated surgery" and wakes up with a hysterectomy may not feel kindly toward her surgeon. If she sues him, the chances are that the jury will decide whether or not she was duly warned in advance and consented to the hysterectomy. The jury may very well believe the patient instead of the doctor, since it is his word against hers.

STATUTE OF LIMITATIONS

Statutes of limitations are laws which state the time during which a person must initiate a suit after the incident before the right to start the suit is lost because of procrastination. In most states, this period is from 1 to 3 years for malpractice, but is longer in several states.

In cases of minors or mental incompetents, the statute of limitations does not become effective until the minor is of legal age, or the mental incompetent becomes mentally competent. Assuming a 3-year statute of limitations for malpractice, it is theoretically possible that if a needle is broken off in a newborn, he may sue the physician responsible almost 24 years later, since the statute of limitations does not become effective until he is 21 years of age. The time varies from state to state, but, at any rate, it is a long, long time.

How many practicing surgeons know who carried their malpractice insurance 20 years ago. Possibly a small minority do. Save old insurance policies. The responsible carrier is the one insuring at the time that the malpractice occurred.

Also, some state courts have held that the statute of limitations does not become effective until the patient learns of the malpractice. Therefore, if a sponge is left in an abdomen and not removed until 10 years later, the statute becomes effective when the sponge is removed.

Also, it is generally held that if the surgeon withholds information from his patient, when such information concerns an act of his which is malpractice, such as may occur under anesthesia without the patient's knowledge, he is guilty of fraud, and the statute does not become effective until the patient learns of it.

MEDICAL RECORDS

Brief medical courses covering basic medical subjects are taught in all better law schools. Many trial lawyers spend many hours studying medicine and surgery and pride themselves on their medical knowledge, which may be quite extensive.

The medical record is available to these legal experts with medical knowledge. The hospital may refuse to give it to the lawyer who has the consent of the patient to release the medical information, but he can always subpoena it and examine it in great detail. Therefore one should be careful what he writes in a medical record. He may be faced with his statements on a witness stand.

One should avoid derogatory comments about the patient in the record. These discredit the doctor in the eyes of the jury and are ill advised in any case.

When something goes wrong, as inevitably happens, describe the incident in the most favorable light. If one breaks off a needle and is unable to find it, he should note in the operative report that a long and thorough, but unsuccessful effort was made. Or one can make a defense to the effect that the condition of the patient was such that he could not make an adequate search.

If the incident is so blatantly negligent that one has no defense at all, it probably is better to note the incident in the record in a brief manner. Negligence should never be admitted nor emphasized in the written record. On the other hand, one must never falsify a medical record—just present the situation in its most favorable light or mention it in a brief and casual form, so as to minimize the legal problems involved.

CAUSES FOR MALPRACTICE SUITS

Sandor[3] reviewed the types of causes of malpractice actions in some 1936 published legal cases which were appealed to the higher courts between 1794 and 1956. Many thousands of others did not appear, as they were settled out of court or not were appealed.

He found that failure to take x-rays in a suspected fracture was the leading cause of the claims. This certainly is preventable.

Other causes mentioned by Sandor[3] or found in more recent literature are:

Foreign bodies left in operative field (sponges, needles, hemostats, broken glass from suction tips, etc.)
Defective operating room equipment or grounding causing anesthesia explosions
Burns (from hot water bottles, x-ray or radium, diathermy and electrosurgical units)
Chemical burns (from skin prep solutions)
Operating on wrong side
Operating on wrong person
Failure to complete an operation
Lost drains
Lost intratracheal tube
Broken teeth during intubation, causing lung abscesses
Bridgework not removed before operation and swallowed or inspirated during anesthesia
Wrong diagnosis due to failure to perform usual diagnostic tests
Injections of injurious substance into tissues, with a slough, or into the spinal canal or abdominal aorta, with resultant paraplegia

Certainly all of these are preventable.

Blood transfusion[1] with incompatible blood happens much more frequently than is generally recognized, particularly under anesthesia. Mortality from blood transfusion is estimated at one death in 1000 to 3000 transfusions. The Veterans Administration requires elaborate precautions in an attempt to prevent these accidents, but still an occasional case occurs.

A frequent cause which must be read into the case is faulty positioning on the operating table, or pulling too hard on retractors. These would explain a number of postoperative peripheral nerve palsies, most frequently brachial plexus or peroneal nerve. Practially all of these are preventable with due care. The indicated corrective actions are obvious.

RESPONDEAT SUPERIOR

Respondeat superior is the legal doctrine by which a physician assumes legal responsibilities for the acts of persons working for him when they perform for him in a medical capacity.

This ordinarily includes office staff, internes, residents, nurse anesthetists, and some of the hospital nurses. Therefore, one should be careful to delegate only such duties as are customarily performed in his or a similar community and which the staff is trained to do.

A physician is likewise liable for negligence of his partners.

RES IPSA LOQUITUR

Res ipsa loquitur, or "the thing speaks for itself," is proof of negligence by circumstantial evidence. It has probably developed because of the difficulty of getting one doctor to testify

against another, particularly as an expert witness.

Ordinarily when one person sues another for an injury, he must prove that the other person caused the injury because of negligence. Under the doctrine of res ipsa loquitur, the shoe is on the other foot. The physician being sued is assumed to be guilty and must prove that he is not guilty.

For example, an anesthetized patient falls to the floor, breaking a bone, while being lifted from the operating table to a stretcher. The patient has no way of knowing what happened and perhaps could not get those present to testify in his behalf. Here, under res ipsa loquitur, the responsible surgeon and others participating would be held liable for the accident, unless they could prove that the accident happened in such a way that they were not negligent.

Adoption of res ipsa loquitur varies from state to state, but it is becoming increasingly popular with the courts.

FEES

If the patient does not make some effort to pay after several gentle reminders, proceed cautiously. Malpractice suits are a common form of counterattack when disgruntled patients are pressed for fees. Even if the suit is won and the physician exonerated, such suits are very poor publicity.

In many states a lawsuit for malpractice is outlawed by the statute of limitations several years before the bill is. In such cases, the physician should merely wait until the time for the possibility of a malpractice action expires, and then become more vigorous in his collecting.

Also, it is wise not to fire an employee who has personal knowledge of a malpractice situation until the suit is over. An angry employee makes an excellent witness for the opposition.

SUMMARY

A few of the aspects of malpractice suits have been discussed from a surgical, rather than a legal viewpoint.

Most malpractice suits are preventable with due care and proper physician-patient relationship.

Increased training of surgical residents in the fields most likely to produce malpractice suits should help prevent the suits.

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MESENTERIC THROMBOSIS: DIAGNOSTIC SUGGESTIONS*

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Fortunately, mesenteric thrombosis is a relatively uncommon disease. Unfortunately, this does not allow the average surgeon to accumulate as much experience as he needs in handling the condition. Several excellent clinical descriptions have been given in the past medical literature,[1, 4, 5, 7] but it nevertheless remains a formidable diagnostic and therapeutic problem. Despite tremendous advances in surgical techniques and methods, the mortality rate remains a striking 70 to 80 per cent.[1] This is probably due largely to the very nature of the pathologic process involved; however, it is also due in part to the difficulties of diagnosis. In its full blown state, it has been termed the most catastrophic of all abdominal emergencies, but there exists another form of the disease that is very insidious and beguiling. It has been appropriately called the masquerader of the abdomen, for it is most difficult to identify.

It is the purpose of this report to emphasize this latter aspect of the disease which is all too often not recognized because physicians often think only in terms of the more dramatic form. Two illustrative cases are cited. From a careful analysis of these cases it is believed that certain practical precepts may be set forth that will aid the clinician in reaching an early decision. Surgical intervention remains the only successful form of treatment. Most of these precepts, it must be added, have been gained in retrospect and from collateral study of the problem and were not necessarily applied in the handling of these cases. It was failure to use them that has highlighted this problem.

CASE REPORTS

Case 1. J. H., 50-year-old White Man

(*MBH 72-197*)

Chief complaint. Abdominal pain.

Present illness. Five days before admission the patient had a sudden onset of abdominal pain while in church. On the following 2 days there was slight bloody mucoid diarrhea; he had a normal bowel movement the next day and none on the day of admission. The pain persisted as a constant symptom, located in the midabdomen and epigastric areas with extension through to the back and with considerable cramping at times. There was slight nausea but no vomiting. He was admitted to the hospital on May 29, 1959.

Past history. The patient had had pulmonary tuberculosis 18 years earlier, for which he had received a right thoracoplasty. Ten years earlier an appendectomy had been done and 3 years before admission, a cholecystectomy. For 3 months before admission, he had "prostate trouble" requiring frequent administration of antibiotics. The last dose of antibiotics was taken on Friday before onset of present illness on Sunday.

Physical examination. Blood pressure, 150/70; pulse, 88 per minute, respiration, 22 per minute; temperature, 99.8 degrees. The patient was a thin white man sitting up in bed in moderate pain. There was slight fullness of the abdomen with resistance to pressure in the upper abdomen. He had tenderness over the entire epigastrium. The lower half of the abdomen was less tender and less resistant to pressure. No active peristalsis could be heard. There were right upper and lower abdominal scars. No hernias were noted.

Laboratory studies.

X-rays: Flat and erect plates of the abdomen revealed no free air and no evidence of bowel obstruction.

Complete blood count (CBC): hemoglobin, 16.8 gm.; hematocrit, 51 volume per cent; white blood cell count (WBC), 7400 (segmented neutrophils (segs) 68, eosinophils (eos) 1, lymphocytes (lymphs) 31, monocytes (monos) 1).

Urinalysis: normal.

Serum amylase: 152 units (normal 40 to 110).

EKG: normal.

Clinical Course. The patient continued to have severe abdominal pain relieved only by narcotics; it was somewhat cramping but with a constant character. It subsided enough to allow for colon x-ray on the 3rd hospital day. This revealed diverticulosis but no evidence of diverticulitis. Based on the severe pain and the slightly elevated serum amylase level, a working diagnosis of

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pancreatitis was made. The patient appeared to improve, and on the 4th hospital day, peristaltic activity was audible. His temperature remained normal, and repeated white blood counts were normal. On the 5th hospital day there recurred an acute severe unremitting abdominal pain and again there was marked tenderness in the upper abdomen. The blood pressure rose to 160/120. A Levin tube placed in the stomach yielded a small amount of bright blood. A repeat serum amylase determination was 128 units (normal, 40 to 110); WBC was 27,800 (segs 86, lymphs 12, monos 2); Hb, 17.3 gm.

A decision was reached to undertake surgical exploration of the abdomen, but before this could be commenced, the patient's condition deteriorated further. The pulse rate went to 190 per minute and the blood pressure became unobtainable. EKG revealed ventricular tachycardia with a variable rate 160 to 190 per minute. The patient's blood pressure was partially maintained with continuous Levophed infusions, but his condition continued to deteriorate and he died on the 6th hospital day.

Postmortem findings. The abdomen contained about 1500 ml. of watery blood-tinged fluid. The small intestine showed black hemorrhagic discoloration with infarction extending from a point 14 inches below the ligament of Treitz and measuring about 4 feet in length. The portal vein, splenic vein, superior mesenteric vein and the small veins arising from it were all distended and occluded by recent thrombus. The pancreas and other organs showed no significant change.

Case 2. J. M. H., 53-year-old White Man

(UMC 23238)

Chief complaint. Abdominal pain and vomiting blood.

Present illness. The patient came to the hospital emergency room at 2:30 a.m., September 26, 1959, with the above complaint. One week previously he had developed periumbilical abdominal pain relieved only by codeine. Two days before admission he had had a similar episode, and only 7 hours before admission he had had another sudden onset of periumbilical pain of a cramping character. It was followed in about 2 hours by vomiting, the last two specimens containing dark blood. The pain persisted and was somewhat relieved by a narcotic injection.

Past history. For about 5 years before this he had been under active treatment for rheumatoid arthritis, taking high dosages of salicylates and later steroids. He had developed an ulcer of the lesser curvature of the stomach while on this therapy, and two years before this admission he had submitted to a gastric resection, a Billroth I procedure being performed. After that he had been free from abdominal symptoms until the present illness. His antirheumatic therapy had been continued up to the time of the present illness and recently consisted of gold thiosulfate, Plaquenil and Medrol.

Physical examination. The patient appeared to be thin and chronically ill with moderate abdominal discomfort. Blood pressure, 104/70; pulse, 72 per minute; respiration, 16; temperature, 98.6 degrees. Examination of the abdomen revealed moderate tenderness in the periumbilical region with slight muscle guarding. No masses could be felt. Peristalsis was audible and of normal quality. Well healed abdominal incisions were noted.

Laboratory studies.

X-rays: Flat and erect plate revealed no evidence of free air and no evidence of bowel distention. Upper gastrointestinal series revealed no evidence of esophageal varices and no evidence of ulceration of the stomach or anastomotic site.

CBC-Hb.: 11.3 gm., 72.9 per cent; hematocrit, 36 per cent; WBC, 7600 (segs 62, bands 7, metamyelocyte 1, lymphs 27, eos 3).

Urinalysis: normal.

Serum amylase: 50 units.

EKG: normal.

Hospital course. While under observation the patient continued to have intermittent abdominal cramping pain and several times vomited additional small quantities of blood. His abdomen remained slightly tender but not rigid. Bowel sounds continued to be audible. It was thought that the patient probably had a penetrating bleeding ulcer, and he was treated with blood transfusion and followed closely. Approximately 24 hours after admission, the blood pressure fell to 78/60, and there was increasing abdominal pain with greater rigidity and a disappearance of bowel sounds. He was explored some 26 hours after admission.

Operative findings. The abdomen contained approximately 1 L. of foul smelling dark maroon colored fluid. Black distended bowel which was grossly gangrenous was noted from approximately 10 inches below the ligament of Treitz to within 1 inch of the ileocecal valve. The mesentery was thickened and edematous and without pulsations. This entire segment of infarcted small bowel was resected, consisting of approximately 8 to 10 feet (pathologist's estimate); direct continuity was re-established, leaving approximately 1 foot of small bowel between the ligament of Treitz and the ileocecal valve. Pathologic examination of the surgical speciman failed to reveal whether the thrombus was primarily venous or arterial.

Further course. Postoperatively, despite a rather stormy course, the patient recovered satisfactorily, and was discharged on the 18th postoperative day. He was required to restrict himself to a bland diet and had five or six loose stools a day. His arthritis continued to be a problem, and steroid therapy was quite difficult to maintain because of electrolyte problems. He was re-admitted to the hospital twice in the next few months in coma with serious electrolyte imbalance and malnutrition. Five months after surgery the patient was re-admitted for the fourth and final time and died from bronchial pneumonia and septicemia. Despite intensive efforts at nutritional maintenance he had shown marked emaciation. Autopsy revealed the length of the small bowel from the ligament of Treitz to the ileocecal valve to measure 12 inches. There was no evidence of further thrombosis of the mesentery or ulceration of the bowel.

DISCUSSION OF CASES

It will immediately be noted that there is a striking difference between these two cases and the usual dramatic form of the disease in which the onset of symptoms is abrupt and fulminating and the disease progresses within the matter of a few hours to a full climax. In these more typical cases, the patient is seen prostrated with pain and probably in shock. Physical signs of a severe peritoneal insult are usually unmistakable to the surgeon, and there is usually no great cause for hesitation in operating on such patients. The high mortality rate in such cases can be attributed not so much to delay in diagnosis as to the extent of disease encountered.

In the two cases presented, by contrast, we find a disease process that is developing in a much slower manner with a clinical course extending over several days. In both cases, mesenteric thrombosis was determined to be the sole cause of abdominal symptoms; this was proved by surgery in one case and by post-mortem examination in the other. It must be concluded, therefore, that the pathologic process began as a small thrombosis and progressed by slow propagation of the thrombus or by a series of repeated small thrombotic episodes until the fully developed pathologic condition was reached. This inference seems verified by the history in each case of repeated attacks of cramping pain that would be interrupted by seeming improvement for short intervals. The tenderness and the cramping pain which were present during these acute episodes indicated partial ischemia; however, complete disappearance of peristaltic activity and the signs of peritonitis that accompany infarction did not occur until the final stages of the disease. It is to be deduced, therefore, that had surgical exploration been carried out in either of the two cases at an earlier stage, a much more limited process would have been encountered, which might have resulted in an altogether different outcome.

The slowly progressive form of the disease is more probably related to venous thrombosis than to arterial occlusion; however, this is not definitely established, and it is entirely possible that a small arterial occlusion might incite the process and lead to retrograde thrombosis of the veins and from the area involved. Clinically, it is almost impossible to distinguish whether vascular occlusion is on an arterial or venous basis. One might suspect arterial causes when there is associated cardiovascular disease, and venous causes when there is a previous history of venous thrombosis or when there is associated visceral inflammatory or traumatic disease. Regardless of the process, the cases have similar clinical patterns, and it is believed that those cases in which the course is by slow degrees over a period of several days represent ideal cases for arresting the disease in the early stages when they may be most successfully treated. This emphasizes the necessity of early diagnosis in this the insidious or gradual form of the disease. After reviewing the problem carefully and especially the clinical findings in the above two cases the following diagnostic points are set forth:

1. The fact that mesenteric thrombosis does occur in this slower milder form must be kept in mind. The symptoms and signs are likely to be atypical, and several other disease possibilities must undoubtedly be ruled out in differential diagnosis. When adjunctive laboratory studies do not strongly confirm other diagnoses and one is resting on a weak or doubtful working diagnosis, there must be a surgical readiness to carry out exploration at any time that the patient does not respond or seems to deteriorate. Exploration will usually be safer and more satisfactory than procrastination.

2. Pain in each case was out of proportion to the obvious clinical findings. This is a symptom that is not measurable and admittedly subject to marked individual variation; however, several

observers on each case were similarly impressed. It is believed, therefore, that a valid precept might be set forth that in such cases that fall into this general category of suspicion, the continued presence of manifestations of severe pain without entirely adequate explanation might in itself constitute sufficient justification for abdominal exploration. A short period of time must be taken, of course, to evaluate the persistence and severity of the pain and the patient's subjective reliability. This may also be used to rule out those conditions which also present the pain picture but which have measurable diagnostic criteria such as pancreatitis, coronary disease, etc. Nevertheless, when these have been considered and found untenable and the patient still exhibits fairly severe or persistent pain, exploration should be considered far safer than "conservative" observation.

3. The presence of blood in some form in the gastrointestinal tract may be looked on as a clue that will heighten one's index of suspicion in cases such as these. In one case there was a bloody mucoid diarrhea which was thought to be due to the recent intake of "mycin" drugs. In the second case there was bloody vomitus that was thought to be due to marginal ulcer. Although plausible, the first conjecture was not altogether in keeping with the working diagnosis of pancreatitis; and in the second case, the marginal ulcer was not demonstrated by an upper gastrointestinal x-ray study. In both cases there was undoubtedly a bloody effusion into the bowel from a small infarcted segment. There are many other causes of gastrointestinal bleeding, of course, but this finding coupled with that noted above should cause one to consider more strongly an earlier surgical procedure.

4. A diagnostic peritoneal aspiration was not done in either of these two cases. It is possible that the diagnosis might have been clearer had this been done. The use of this technique as a diagnostic tool is discussed well by Gray and Amador[2] who indicate that it is an excellent way to differentiate between pancreatitis and infarcted bowel, two of the conditions that closely simulate one another in the "acute abdomen" classification. Bloody ascitic fluid is present in both, but with infarcted bowel it has a foul odor and shows the presence of *Escherichia coli* on smear. Hill,[3] in experimental dogs, has revealed that these bacilli appear in the peritoneal fluid within 4 hours of establishing infarction. Jenson and Smith[4] have suggested a modified technique of aspiration by inserting a polyethylene tube through a no. 13 needle; this, they contend, will detect even small amounts of ascitic fluid when ordinary methods fail. This procedure deserves widespread clinical trial.

5. Aortography to evaluate the status of the celiac and mesenteric arteries may prove valuable. If mesenteric arterial occlusion is demonstrated, corrective arterial surgery is indicated.

SUMMARY

Two cases of proved mesenteric thrombosis are presented, each of which was clinically slow and insidious in onset, presenting much difficulty in diagnosis. This form of the disease is not commonly recognized, and it is therefore emphasized as a clinical entity to be borne in mind by the surgeon. Practical precepts to aid in its early diagnosis are elaborated. Aortography should be employed earlier and more frequently than at present in cases of suspected mesenteric arterial occlusion.

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ADDENDUM

Since this manuscript was completed, a case of superior mesenteric arterial occlusive disease has been diagnosed by aortography and correction achieved by Teflon bypass (Drs. Watts R. Webb and James D. Hardy).

THE MANAGEMENT OF ACUTE TRAUMATIC DISLOCATIONS IN ADULTS*

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This presentation deals with acute traumatic dislocations of the upper and lower extremities. No attempt is made to include all types of injuries; only the more frequent problems will be discussed.

In the management of acute dislocations the following are imperative: (1) early recognition of the injury, (2) realization of immediate or delayed complications, (3) adequate pre- and postoperative care, (4) prompt reduction, and (5) restoration to previous function without prolonging the patient's morbidity.

Classification into partial or total displacement is useful, but may lead to confusion. As with fractures, the dislocation may be closed or open. A closed dislocation does not imply that initial damage to the joint, soft tissues, blood vessels, nerves and bone is inconsequential. In a fresh dislocation, forceful separation of the articulating bone from contact with its articular surface causes a total or partial rupture of the joint capsule, permitting passage of the dislocated bone, extra-articular or intra-articular ligamentous tears, cartilaginous fragmentation and pitting, tendinous, muscular, periosteal and bony damage.[40]

A concise history (where, when and how), a thorough physical examination and the indicated roentgenograms lead to prompt diagnosis. Neurologic and vascular evaluations of the involved extremity, recorded in detail on the patient's chart, are a must before and after reduction. *The treatment of all dislocations is immediate reduction; delayed care makes relocation difficult, enhances complications.* When the patient is first seen, the suspected area of dislocation is immobilized with a splint, sling, or whatever material is available to prevent further soft tissue damage and blood vessel and nerve injury. After premedications, atropine, or scopolamine and a narcotic have been administered, general or conduction anesthesia[37] is required. (Exceptions are phalangeal displacements when local anesthesia is used or employment of the gravity method when only narcotics are given.) The most gentle but effective reduction procedure should be tried first. Pump handle action or rapid manipulation before muscular relaxation leads to difficulty in relocation and further muscular, capsular and bony damage. The extremity is immobilized with a suitable dressing, splint, or circular cast for from 4 to 8 weeks. Obviously, the time factor in immobilization depends on the extent and location of injury, the age of the patient, and the experience of the surgeon. Postreduction roentgenograms show the accuracy of joint replacement and note any bony complications which may occur during reduction. When the immobilizing agent is removed, supervised graduated active and resistive exercises are instituted. Forced passive motion and manipulation will add to postreduction complications such as myositis ossificans or traumatic arthritis. Excessive motions of the involved joint are avoided for 6 to 8 weeks, after removal of the immobilizing agent.

Conservative management of traumatic dislocations produces the best results. *Immediate surgical intervention in the uncomplicated dislocation is not necessary and may mean that the surgeon has operated needlessly.*

Immediate surgical intervention is required in an open joint injury, in vascular or neurologic complications, and tendinous, bony or muscular tissue interposition preventing normal joint relocation. *Surgical care of open dislocation depends on basic surgical skill, not on antibiotic coverage.* The involved joint is contaminated, and the tragic end results of infection may develop despite prompt treatment. After the open wound is primarily debrided and irrigated with 5000 to 7000 ml. of isotonic saline, and after gowns, drapes and gloves have been changed, a secondary debridement with separate instruments is carried out. This consumes time, but it

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is far less troublesome than months of hospitalization spent by the patient with a joint infection. With adequate debridement, most open dislocations may be closed primarily. If the wound is grossly contaminated, the joint capsule must be closed, but soft tissue may be left open and sterile dressings applied. The choice of primary or secondary closure of the soft parts and skin 3 to 5 days after initial surgery must be the surgeon's decision. There can be no rigid routine, but each case should be judged separately on the basis of the surgeon's experience and training. Medication includes injection of an antibiotic into the joint after closure, a booster of 0.5 ml of tetanus toxoid or 5000 units of tetanus antitoxin (after negative skin test), and a broad spectrum antibiotic.

In a chronic dislocation less than 3 weeks old, gentle manipulation under adequate anesthesia can first be attempted. If this proves ineffectual, an open reduction with full realization of the technical difficulty is done. A perfect reduction in a closed dislocation does not guarantee an uncomplicated end result. Delayed stigma, traumatic arthritis, aseptic necrosis, recurrent dislocation, myositis ossificans, Sudeck's atrophy, ossifying hematoma and decrease in range of joint motion may occur.

UPPER EXTREMITY

Shoulder

The shoulder,[29, 113] sheltered by a shallow concave glenoid fossa and acromial roof, bears the brunt of trauma in people between 18 and 40 and accounts for over 50 per cent[39] of all dislocations. The loose articular capsule, coraco and glenohumeral ligaments, glenoid labrum, articulating cartilage of the humerus, and glenoid are damaged to varying degrees.[5, 23, 78]

Anterior dislocations. The types[93] of anterior shoulder dislocations are: (1) subcoracoid (common), (2) subglenoid, (3) subclavicular, (4) supracoracoid, and (5) supraglenoid. A blow to the externally rotated and abducted shoulder will cause an anterior dislocation.[35, 97] The symptoms are a painful, limp extremity with depression in the subacromial area and a palpable anteromedial humeral head in the pectoralis mass. Roentgenograms in the anteroposterior and transthoracic view confirm the clinical impression and detect any associated humeral, scapular and clavicular fractures.

Treatment.

1. Gravity and weight (gentle) requires no anesthesia other than sedation or a narcotic. This is effective in 80 to 90 per cent of the cases in which it is used. With the patient prone,[127] the dislocated arm is placed over the side of a table with 5 or 10 pounds weight for 10 minutes. The gradual pull of the weight relaxing the shoulder muscles will reduce the dislocation.[108]

2. The Hippocratic maneuver (simple) requires a general anesthetic. The unshod foot is placed in the axilla, the patient's wrist is grasped and gentle traction in the abducted position in line with the long axis of the humerus is carried out.

3. The Kocher maneuver (traumatizing[79, 100]) is a lever force transmitted with external rotation. The forward adduction and internal rotation to the humeral head can result in fracture of the humerus, subscapularis tear and injury to the axillary nerve and vessels. It must not be used indiscriminately or by the uninitiated.

After reduction, a Velpeau dressing, with the hand and wrist visible, is applied for 3 weeks to allow for capsular and soft tissue healing. Longer immobilization will lead to a stiff shoulder, especially in the older patient, and does not decrease the incidence of recurrent dislocation. Circumduction and pendulum movements[38] to the limit of pain are begun, graduating to resistive exercises. Overhead motion and hyperabduction are avoided for 2 months.

Irreducible, acute dislocations due to capsular invagination, interposition of the rotator cuff or bony fragment, and posteriorly placed long biceps tendon will require open reduction.

In an unreduced dislocation,[9] 4 weeks or more, one attempt at closed reduction, using the Hippocratic maneuver, is made. If reduction is not possible, open surgery[25] is necessary. *The end result does not insure normal motion.* In an elderly person, it might be best to leave the displacement alone and accept the disability.

Habitual dislocations[103] in an active person[52] can occur despite prompt reduction and adequate care. A single recurrent dislocation of the shoulder does not mean mandatory surgery. Several of many procedures may be used: (1) repair of the torn anterior capsule (Bankhart),[14] (2) plication of the subscapularis and capsule (Putti-Platt),[16] (3) lateral displacement of the subscapularis tendon (Magnuson-Stack),[46] and (4) anterior bone block (Eden-Hybbinette).[33, 54]

The best and most logical approach is to repair whatever the primary defect may be at the time of surgery.

Posterior dislocation. The posterior dislocation caused by trauma to the adducted and internally rotated extremity is often undiagnosed. It is subacromial or subspinous with a palpable posterior humeral head, internal rotation of the humerus, marked limitation in abduction, and a prominent coracoid. An axillary or tangential[98, 131] roentgenogram obtained at the time of injury is an invaluable aid. Use of the Hippocratic maneuver with a push against the humeral head from behind affords reduction. Velpeau immobilization for 3 weeks is necessary; active and resistive exercises can then be instituted. Maintenance of reduction may be difficult.[31, 81] Rarely, a threaded pin is inserted to fix the head to the glenoid to maintain stability.[128] The pin is removed in 3 weeks.

In recurrent or unreduced dislocations, a reverse Putti-Platt[106] subscapularis transposition[71] or Bankhart procedure is done. *The most serious complication is failure to make a correct initial diagnosis.*

Complications of acute shoulder dislocations are: (1) damage to the brachial plexus, often the axillary nerve, (2) fractures of the surgical or anatomical neck, greater tuberosity of the humerus, scapula or clavicle, (3) recurrent dislocation, (4) brachial vessel damage, (5) deltoid atrophy, (6) limitation of motion, (7) pain, and (8) traumatic arthritis.

Acromioclavicular dislocation. The subcutaneous acromioclavicular joint is commonly displaced superiorly by a direct blow to the lateral aspect of the shoulder.[122] In an incomplete dislocation, the acromioclavicular ligament is torn, but in a complete injury the coracoclavicular and acromioclavicular ligaments are ruptured.[123] Marked elevation and pain are present. Postero-anterior roentgenograms confirm the diagnosis.[20, 117]

Incomplete separation is treated with a posterior figure-of-8 dressing or a compression sling which exerts a downward pull on the clavicle and an upward push on the acromion. Local anesthesia injected into the displacement relieves discomfort. In a complete separation, a compression sling can be used. Rarely, because of complications which arise,[76] transfixion of the acromioclavicular joint with threaded pins and repair of the coracoacromial and acromioclavicular ligaments may be necessary.[10, 50, 80, 90]

In an unreduced dislocation with pain, treatment is excision of the lateral 3 cm. of the clavicle.[47, 77] *A mild, painless displacement does not warrant an operative procedure.*

Sternoclavicular dislocation. Sternoclavicular dislocations are classified as: (1) anterior ((a) superior, (b) inferior) and (2) posterior. An antero-inferior displacement is the most common. Incomplete displacements are often overlooked.[69] In the obvious anterior dislocation, local procaine is injected into the sternoclavicular joint and traction is applied to the abducted arm with digital pressure directed over the displaced fragment. An anterior figure-of-8 dressing reinforced by adhesive straps across both sternoclavicular joints is applied for 4 weeks. An alternate method is to use lateral traction for 4 weeks with a sandbag over the displaced area; this requires hospitalization.

In a posterior dislocation,[115] a surgical emergency may be present; the trachea, esophagus or superior vena cava can be ruptured. Reduction of the uncomplicated posterior dislocation is achieved by posterior displacement of the outer clavicle levering the inner displaced portion anteriorly. A posterior figure-of-8 dressing is applied for 4 weeks. When adequate reduction cannot be obtained by conservative measures, digital reduction and threaded pin fixation are performed. A posterior figure-of-8 dressing is applied for 4 weeks. If the intra-articular cartilage prevents closed reduction, disc replacement and repair of the sternoclavicular ligament are necessary.

In a chronically displaced anterior or posterior sternoclavicular joint,[32] arthroplasty is seldom successful. The most simple,, effective method of relieving discomfort is excision of the medial 2 cm. of the clavicle.[106]

Elbow Dislocation

An elbow dislocation may be: (1) posterior (common), (2) anterior, (3) lateral and (4) medial. The posterior dislocation creates rapid swelling in the antecubital space with a varus or valgus deformity, prominent olecranon and a shortened extremity.[129] A general anesthesia or brachial plexus block is done; the forearm is displaced posteriorly, releasing the coronoid impinging on the posterior humerus. Anterior

pressure is exerted on the humerus and the forearm is brought forward and flexed. Full extension of the elbow is not necessary; if this is done, damage to the brachialis muscle or anterior capsule, leading to myositis ossificans, may occur.[112] Immobilization is maintained by a posterior splint with 70 to 90 degrees of elbow flexion and supination for 3 weeks. Early active exercises are important to regain elbow motion. The patient should not be allowed to carry excessive weights or passively to stretch his elbow. Complications are: (1) limitation of joint motion, (2) traumatic arthritis, (3) myositis ossificans, (4) instability, (5) fractures of the ulna, radius or humerus, (6) pain, and (7) vascular or nerve damage.[22, 34, 55, 58] In an unreduced dislocation 2 weeks old, if an attempt at closed reduction is not successful, open surgery is necessary.[107, 124]

In a Monteggia fracture, closed reduction is done with digital pressure exerted on the radial head and the elbow placed in full supination. Immobilization in 70 to 90 degrees of elbow flexion and supination in a posterior splint is necessary for 6 to 9 weeks. If reduction fails, surgery is done, with reduction of the ulna fragment by internal fixation and replacement or removal of the radial head.[24, 87] With surgery, early motion (3 weeks) is possible. In recurrent dislocations of the elbow, surgery may be necessary.[62]

Distal radioulnar dislocations. After a fall on the pronated hand, ruptures of the radioulnar and ulnar collateral ligaments occur with anterior or posterior displacement of the radioulnar joint. In the anterior dislocation, treatment consists of digital pressure reduction under conduction anesthesia. In a posterior displacement, the ulna is directed anteriorly with digital pressure. Immobilization is in a short arm cast in 5 to 15 degrees of dorsiflexion for 6 weeks. Excision of the distal ½ to ¾ inch of the ulna is done in a chronic painful wrist.[27, 30]

Carpal Dislocations

A fall on the outstretched hand may cause: (1) lunate[49] displacement or (2) perilunar carpal row shift. The common volar injury is the only true lunate displacement. The lunate is pushed forward and is palpable under the flexor tendons and volar carpal ligament. There is limitation of joint movement, pain and swelling. If the injury has occurred within 3 weeks, closed reduction is attempted under conduction or general anesthesia. With the wrist extended, the thumb is placed on the palpable lunate, pressure is exerted, the wrist is flexed and relocation may be effected. A circular cast, with the wrist in 15 to 20 degrees dorsiflexion for 4 weeks, is necessary. Postreduction pain may be caused by traumatic arthritis or aseptic necrosis (Kienbock's disease).[61] The carpal row shift[66] finds the lunate in place, the posterior edge of the capitate palpable, and the carpal row posteriorly displaced. This is reduced by traction on the hand with thumb pressure exerted over the dorsum of the wrist, under conduction or general anesthesia. The wrist is placed in a circular cast in neutral or 10 degrees dorsiflexion for 4 weeks. In chronic lunate dislocations, a disability is present. When roentgenograms show viable bone, open reduction is done.[28] Lunate removal is indicated 6 months after injury or if aseptic necrosis with pain is present. In a chronic perilunar dislocation, open reduction is necessary. Occasionally a wrist fusion is indicated in a perilunar dislocation if the articular cartilage of the wrist is damaged so that persistent pain is present and is not relieved by rest, salicylate therapy or intra-articular Depo-Medrol.* Isolated dislocations of the carpal bones, although rare, do occur and should be thought of whenever there is a traumatic injury to the hand.[18, 44, 57, 64, 89, 125]

Radiocarpal dislocations. This injury, the result of extensive trauma to the wrist, often open, is accompanied by fractures of the distal radius and ulna. The dorsal deformity is prominent. Reduction is under general or conduction anesthesia with relocation of the deformity. A long arm cast or posterior splint is applied for 6 weeks. The elderly patient must begin pendulum and circumduction exercises to the shoulder to avoid proximal joint limitation of motion. A wrist fusion is necessary only if persistent pain is present after reduction.

Carpal metacarpal dislocations. A direct blow on the carpal metacarpal joint results in a posterior or anterior dislocation.[110] The most frequent injury is to the first metacarpal with an accompanying fracture, termed a Bennett fracture.[7] Rarely, a Y fracture is present.[102] In a

* Upjohn trade name for intra-articular methylprednisolone acetate.

Bennett fracture there is pain, swelling and shortening of the involved first digit.[130] Reduction under conduction anesthesia is done and the thumb is placed in an abduction splint for 4 weeks. If reduction is not accomplished, skeletal traction with a threaded pin through the distal tuft or phalanx for 4 weeks is necessary.[11] In dislocations of the second to the fifth carpal metacarpal joints, reduction is achieved under local anesthesia followed by immobilization for 2 to 3 weeks in an anterior and posterior splint. With a chronic Bennett fracture, fusion[17] of the base of the first metacarpal and greater multangular may be necessary.

Metacarpal phalangeal dislocations. The common posterior dislocation of the metacarpal phalangeal joint[52] is treated by digital pressure against the dorsal surface of the base of the proximal phalanx under conduction anesthesia pushing it anteriorly. Traction on the *distal* phalanx is avoided. The palmar ligament will invaginate into the joint space, not allowing a reduction. With interposition of the palmar ligament or volar joint capsule and straddling of the metacarpal head by the thenar muscles, closed reduction is not possible and surgery is necessary.

When either open or closed reduction is accomplished, an anterior plaster splint is applied for 2 to 3 weeks.

Interphalangeal Dislocations

The most common type of interphalangeal dislocation[10] is posterior. Gentle traction, with or without local anesthesia, followed by immobilization in 20 degrees of flexion with an anterior splint for 1 week is sufficient.

LOWER EXTREMITY

Hip

The triaxial joint of the hip is protected by a cavernous, concave, acetabular fossa requiring a severe force to dislocate. The varieties of dislocations are: (1) posterior (common) ((a) iliac, (b) ischial), (2) anterior ((a) obturator, (b) pubic, (c) perineal), and (3) central.

Posterior dislocations. The ischiocapsular ligament, vulnerable to a blow transmitted through the lower femur when the hip is flexed, adducted and internally rotated, is torn. The forcible collision[41] causes a posterior capsular rent and ligamentum teres or fovea capitis injury with fractures of the posterior labrum and/or femur.[19] The hip remains flexed, adducted, and internally rotates[42] with prominence of the greater trochanter, leg shortening and pain.[116]

Treatment.

1. The Stimson gravity method[72] (gentle) requires only a narcotic. This is successful in 70 to 80 per cent of the cases in which it is used. Weight of patient's extremity, aided by downward traction on the popliteal space, reduces the displacement.

2. The Allis method (uncomplicated) requires conductive or general anesthesia and direct upward traction on the flexed hip.

3. The Bigelow maneuver (traumatizing) is used only if the above measures have failed. Conductive or general anesthesia is used. The hip is then flexed, the thigh adducted and rotated internally and externally.

In the postreduction care, straight Buck's traction for 4 to 6 weeks is necessary. Immobilization in a 1½ spica cast may be substituted. The patient can be on light weight bearing 4 to 6 months after injury. If he develops pain (early sign of aseptic necrosis), motion and weight bearing are restricted. Reconstructive surgery (cup arthroplasty), replacement prosthesis and arthrodesis may be necessary.

Anterior dislocation. The Y ligament of Bigelow is not completely torn, rather the head is ejected distally to it when the hip is abducted and a direct or indirect force applied. The limb is reduced under conduction or general anesthesia by upward traction on the flexed hip followed by adduction and pressure against the femoral head.[67] Postreduction treatment is similar to that used in a posterior dislocation.

Central dislocation. A blow on the greater trochanter or the extended hip can drive the femoral head into the acetabular fossa. There is shortening, the femoral head cannot be palpated, and the greater trochanter is upwardly displaced.[106-108]

Treatment. Minimal displacement requires longitudinal traction (Buck's or Russell's) for 10 to 12 days. Ambulation with crutches and partial weight bearing is carried out in 1½ to 3 months. With a marked amount of displacement, skeletal longitudinal traction is applied for 3 weeks with a lateral swathe or screw inserted into the greater trochanter. When

reduction is not possible, open surgery is advisable.[36, 63, 82, 104] Weight bearing is begun in 3 to 5 months.[6, 91] Roentgenograms can be deceiving; what appears to be a poorly formed acetabulum may contain a serviceable and functional hip. *Do not treat on appearance of x-rays.*

Open reduction in acute dislocations is indicated with: (1) large unattached acetabular fragment,[45] (2) intra-articular fragment,[56] (3) sciatic nerve injury,[53] and (4) capsular or glenoid labrum invagination.[3, 26, 84, 85]

The complications[4] of traumatic dislocations[95] are: (1) fractures ((a) acetabular rim, anterior or posterior, (b) femoral head or neck[50, 83, 88, 94, 99]) (2) aseptic necrosis,[96] (3) sciatic nerve injury, (4) traumatic arthritis, (5) myositis ossificans, (6) limitation of joint motion, (7) damage to femoral vessels or nerve, and (8) intra-articular fragment pre- and postreduction.

Temporary disability for 6 months is common with a dislocated hip; a guarded prognosis to 18 months is necessary.

In an "unreduced" dislocation of 3 weeks, closed reduction under conduction or general anesthesia is done. If unsuccessful, skeletal traction (to stretch the capsule and surrounding structures), open reduction and arthrodesis are necessary.[92, 121] This procedure can be formidable and must not be undertaken without adequate preoperative preparation, operative facilities, personnel and surgical training. Recurrent dislocation, although unusual, does occur.[120]

Knee

The types of knee joint dislocations are: (1) anterior (common), (2) posterior, (3) lateral, (4) medial, (5) rotary and (6) combinations of above.

Severe violence, with rupture of both collateral and cruciate ligaments,[1, 8] results in hemarthrosis and marked soft tissue damage. After roentgenograms,[15, 69] the deformity is corrected manually with the knee flexed at 90 degrees. The extremity is immobilized in a long leg cast with 10 to 15 degrees of knee flexion for 2 to 3 months. Active and quadriceps exercises are begun after cast removal.

Semilunar cartilage tears, fractures of the femoral or tibial articulating surfaces, cruciate and collateral ligamentous rupture, popliteal vessel and nerve damage, external peroneal nerve injury, traumatic arthritis, recurrent dislocation and chronic synovitis can result.[65] A knee support with lateral bars or reconstructive procedures (removal of menisci, repair of cruciate or collateral ligaments) may be necessary.

Patella. The triangular patella, guardian of the knee joint, is dislocated: (1) laterally (common), (2) medially, (3) superiorly and (4) inferiorly[2] with a direct blow or a sudden valgus knee injury. The vastus medialis, capsule and collateral ligaments are injured. The abnormally positioned patella is shown in the anteroposterior view.

A spontaneous reduction may occur, but conduction or general anesthesia is often necessary. The thigh is flexed, and the patella is manually replaced in its normal position. A knee cylinder is necessary for 3 weeks. Recurrent dislocation[43] is treated by reefing the medial capsule, transferring the patellar tendon, with a portion of bone, distomedially into the tibia, and inserting the tendon of the semitendinosus into the proximal patella.[68]

Ankle Dislocations

The ankle, a mechanically effective joint, is rarely dislocated without a fracture of the medial or lateral malleolus.[21, 74, 111] The displacement is: (1) posterior (common), (2) anterior, or (3) superior. In a posterior dislocation, the distal tibia and fibula are anterior with the foot posterior. Treatment is immediate reduction under conduction or general anesthesia with the foot plantar flexed, pulled forward and dorsiflexed.

In an anterior dislocation, the heel is shortened, with the distal tibia and fibula displaced posteriorly and the foot anteriorly. The treatment is immediate reduction under anesthesia with the foot dorsiflexed. In both injuries, the limb is placed in a long leg cast with the foot in neutral position for 6 to 9 weeks.

In a compression injury with a superior dislocation of the ankle, there is rupture of the inferior tibiofibular joint and fracture of the distal tibia, fibula and astragalus. Treatment is immediate reduction under conduction or general anesthesia accomplished by downward traction on the foot with the knee flexed. The leg is immobilized in a long cast with the foot in neutral position for 6 to 9 weeks. Open reduction in an ankle dislocation is warranted if

there is: (1) rupture of the tibial, peroneal, extensor or flexor tendons transversing the ankle joint, (2) intra-articular bone fragment, (3) posterior tibial vessel injury, (4) displaced medial or lateral malleoli after reduction and (5) failure of reduction.

When the ankle remains painful and conservative measures (rest, salicylates, brace with anterior and posterior stops) are not effective, an arthrodesis of the ankle is necessary.

Astragalar dislocations. This unusual injury,[12, 70, 75] resulting from a fall from a height, is often open and complicated by malleoli and os calcis fractures.[73] The astragalus displacement may be: (1) anterior, (2) posterior, (3) anterolateral, (4) anteromedial, (5) posterolateral, (6) posteromedial and (7) rotary. The displaced mass of the talus can be visualized and palpated subcutaneously. Immediate treatment, under general or conduction anesthesia, consists of closed reduction with traction applied on the os calcis with the foot plantar flexed and the knee at 10 degrees of flexion. Skeletal traction, with a pin through the os calcis to obtain greater longitudinal pull, may be used. If closed reduction is not successful, immediate open surgery is necessary to prevent sloughing of the skin over the dislocated astragalus.

After reduction, the leg is placed in a long cast with the foot in neutral position for 6 weeks. Aseptic necrosis may develop with or without surgery and will necessitate further immobilization, or if persistent pain is present, an arthrodesis.

Subastragalar dislocations. Anterior,[48] posterior, medial or lateral displacement[13, 114, 118] of the foot with the astragalus remaining in the mortise[109] can result from a fall or torsion. There is obvious swelling and inability to use the foot; peripheral circulation may be impaired. Immediate reduction under general or conduction anesthesia is essential to preserve the circulation of the foot. Reduction is made with longitudinal traction on the foot and counter traction on the leg in the flexed knee position. Direct pressure with the palm of the hand will move the foot to its normal position. Immobilization is in a short leg cast for 6 weeks.

Foot

Midtarsal dislocations. With a severe fall or torsion injury, the midtarsal joint is completely or incompletely displaced in an anterior, posterior, medial or lateral position. The malleoli remain intact, but there is marked swelling and displacement of the forefoot. Treatment is immediate reduction by manipulation under general or conduction anesthesia with molding of the displaced tarsal bones. A short leg cast is applied for 6 weeks. If pain persists after immobilization and physical therapy, consideration of an arthrodesis in the midtarsal region should be made. Individual tarsal dislocations do occur,[19, 51] replacement is achieved by direct pressure under conduction or general anesthesia and immobilization in a short leg cast for 3 to 4 weeks.

Tarsometatarsal dislocations. This uncommon injury can involve the entire group of metatarsals or a single bone.[86, 105] They may be displaced laterally, posteriorly or medially. The most frequent displacements are those of the first and fifth metatarsals. A fracture may accompany the presenting deformity. Therapy consists of traction under local anesthesia and direct pressure on the displaced bone or bones. A short leg cast is applied for 6 weeks.

Metatarsophalangeal dislocations. These seldom seen injuries,[126] caused by direct trauma, have their seat in the first metatarsophalangeal joint. With local anesthesia, closed reduction by manipulation is performed with replacement of the displaced phalangeal joint. Immobilization may be in a posterior splint with the foot in neutral position for 2 weeks or a short leg cast with a walking caliper.

Phalangeal dislocations. The phalanges of the toes are displaced posteriorly after being struck by a falling object or after "stubbing" the toe. Immediate reduction, under local anesthesia, and traction toward the anterior surface with the thumb and index finger is sufficient to relocate the displacement. The injured toe may be strapped to its adjacent neighbor for 1 week.

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EYE INJURIES: DIAGNOSTIC TIPS IN THE MULTIPLE INJURY PATIENT*

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INSPECTION OF THE EYES

If there is marked lid edema, or hemorrhage into the lids, inspection of the globe can be best accomplished with a small tendon retractor or lid speculum. The important thing to remember is to avoid pressure on the globe, as this may extrude ocular contents should the patient have sustained a lacerating injury of the eye. Care should be taken to avoid abrading the corneal epithelium.

SUPERFICIAL ABRASION OF THE CORNEA

If the eye is red or if the patient complains of pain, Fluorescein solution may be instilled into the eye, or filter paper impregnated with fluorescein may be held in the lower cul-de-sac with the tears of the eye creating fluorescein solution. Any injury or disease that has caused removal of the corneal epithelium will, after a few minutes, stain green. In the past corneal ulcers have most probably, at times, been caused by contaminated fluorescein solution. It is recommended that the fluorescein solution used be one that is made up sterile by the manufacturer, usually marketed in a plastic bottle with a screw-on cap, and care should be taken not to contaminate the tip of the dropper bottle. Or, fluorescein-impregnated filter paper, marketed in a sterile envelope, may be used. The latter can be re-autoclaved should any question of sterility arise. Although the abrasion be superficial, if it invades the substantia-propria of the cornea, permanent decrease in vision, as a result of scar formation which either obstructs the passage of light or causes distortion to the passage of light, may be a permanent result. Such abrasion should be treated by bandaging the eye and applying antibiotic ointment. Contrary to some published reports, it has not been this author's experience that superficial abrasions are prone to heal unsatisfactorily in the presence of an ointment (although it has been seen in some inflammations of the cornea).

SMALL PENETRATING OR SMALL LACERATING INJURIES TO THE GLOBE

In contrast to the larger laceration or ophthalmic penetrations in which diagnosis is obvious, the small penetrating injury may be easily overlooked. One should look for partial prolapse of iris, ciliary body or choroid into the wound (irregularity of the pupil may be the first suggestion of a minimal iris prolapse). If aqueous humor has leaked from the wound, the first indication of penetrating injury may be the difference in depth of the anterior chamber of the eye as compared to the fellow eye. When aqueous humor leaks out, the distance between the posterior surface of the cornea and the anterior surface of the lens will decrease. A flashlight held to the side of the eye illuminating the front surface of the iris makes this depth differentation more easily perceived. I have seen a number of cases in which the physician examining a patient from a car wreck noted the probability of a penetrating injury of the globe on the basis of differences in anterior chamber depth alone, but in which the point of penetration was so small as to not be seen on rather careful inspection with the naked eye. Should a lacerating or penetrating injury be found, the most important single point until the ophthalmologist arrives is to protect the patient against any possibility of pressure on the globe. The patient, by rubbing on the eye through the lid, can easily destroy an eye which might otherwise have been saved. A protective cone can be made from cardboard, old x-ray plates, or one of the metallic shields used regularly after intraocular operations. The cone can be taped to the face.

GLOBE HEMORRHAGE

Hemorrhage within the globe may be the only sign of injury, or may be associated with almost any other injury of the globe. If large, hemorrhages in the anterior chamber will, of course,

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be easily seen. If small, particularly if the blood has clotted, they may mistakenly be confused with pigmented lesions of the iris, or if the patient has been in an upright position, they may have settled out into the lower part of the anterior chamber so that they are not easily noted. The lack of luster of the iris of the injured eye (when compared to the fellow eye), giving the iris a "muddy" look, is probably the most common early sign of a minimal anterior chamber hemorrhage. If the patient has been lying on his back, the anterior surface of the iris is the dependent position to which most of the blood settles. If the patient has been upright, the blood will settle to the inferior aspect of the anterior chamber. Hemorrhages into the vitreous humor are less easily noted, unless ophthalmoscopy is performed. With the ophthalmoscope, little or no fundus detail will be seen, but instead a dark muddy reflex encountered. If the hemorrhage has been extensive, nothing but a black void will be seen. Thus, this lesion is named "eightball eye." Hemorrhages in the anterior vitreous humor can often be seen with the ordinary pen light.

INJURIES TO THE LENS

Any penetrating or lacerating injury to the globe that penetrates the lens will usually cause a secondary cataract. If a cataract begins to form within a few days after injury, the probability of an undiagnosed small penetrating lesion of the globe and lens should first be considered. A case of an injury to the lens easily missed is that of the subluxated (*i.e.*, partially dislocated) lens. Here the supporting ligaments of the lens, zonules, have been torn sufficiently to let the lens move posteriorly into the vitreous humor. The most easily seen indication of a partially or totally dislocated lens is the fact that the iris is tremulous. Normally, the lens being directly behind the iris, the iris is held in place and will not shake with ordinary movements of the eye. However, if the lens is not present in its normal position just behind the iris, but is more posteriorly displaced (partial or total dislocation), the iris is free to shake like a blowing curtain in the course of normal movements of the eye. This is seen normally in patients who have had a cataract extraction. A brief examination of the patient's glasses (if they can be found after an accident) can, usually, easily settle the possibility of the patient having had previous cataract surgery. Nearly all glasses worn after cataract extraction are high plus lenses and upon examination will be noted to be rather thick and to magnify.

DETACHMENT OF THE RETINA

The patient's first complaint in most cases of detachment of the retina will be the spontaneous statement that he seems to have a veil or a curtain over part of the field of vision (inferior detachment produces a curtain that seems to run from above down, inferior retina equals superior field of vision). It should be remembered that the patient's ability to read normally does not in the least rule out the possibility of a retinal detachment. It simply means that if a detachment is present, it is not affecting the macula at the time. Ordinary confrontation fields will generally pick up a large peripheral retinal detachment. Examination with the ophthalmoscope will show the retina to be thrown into elevated folds, generally seen between +6.00 and +12.00 with the ophthalmoscope. It should be remembered that the ophthalmoscopic picture of a retinal detachment is not as shown in most textbooks, since a textbook picture is a composite drawing of many planes of depth as seen with the ophthalmoscope, whereas in the clinical examination one can see only that part of the retina lying in the particular plane for which the ophthalmoscope is set at the time. It should be remembered that the best information is gained ophthalmoscopically by starting with a high plus lens, which focuses in the area of the crystalline lens of the eye, and reducing the high plus numbers one at a time so that one is "cutting" successive planes in deeper layers of the eye until ultimately arriving at the retina. A patient with a retinal detachment, when viewed at several inches from the eye with the ophthalmoscope, will not have the orange retinal reflex seen in a normal patient.

RESUMÉ OF EXAMINATION AND SUGGESTIONS

1. Lids. Examine the lids, and open them without pressure on the globe. Use a speculum if deemed necessary.

2. Cornea. Look for roughness of flashlight reflex and green areas upon fluorescein staining.

3. Depth of the anterior chamber. A shallow anterior chamber usually means leakage of

aqueous humor resulting from a penetrating injury.

4. Irregularity of iris. This may be due to minimal incarceration of iris in a lacerating wound, or it may be due to iridodialysis (tear of iris at its base due to blunt injury).

5. Tremulous iris. This indicates partial or complete rupture of zonules, allowing lens to move posteriorly into vitreous humor (or earlier surgical removal of lens).

6. Muddy reflex of eye. (a) Check in the anterior chamber with flashlight for anterior chamber hemorrhage. (b) Use ophthalmoscope for detecting hemorrhage into the vitreous humor or retinal detachment. If the muddy reflex is seen equally in all quadrants, it generally denotes hemorrhage into the vitreous humor; and if it is seen primarily in one quadrant, retinal detachment. (c) Confrontation fields will be essentially the same in all fields (though perhaps reduced somewhat because of poor acuity) in the case of vitreous hemorrhage, and it will be restricted in one field but normal in unaffected fields in case of retinal detachment.

RECENT PROGRESS IN ANESTHESIOLOGY*

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A number of factors are responsible for the unprecedented rate of progress which has been made in anesthesiology during the past decade. As mentioned by Dripps and Dumke in an article published in 1947,[4] increasing recognition of this field as an important medical specialty has perhaps been the most outstanding reason for its rapid growth and development. As a result of such recognition, more and more physicians are entering the specialty training programs in anesthesiology; communication and cooperative effort among clinical colleagues has improved tremendously; and there has been greater interest among basic scientists in helping to solve many of the perplexing problems which are faced daily in clinical practice.

In reviewing the significant contributions in anesthesiology during the past few years, the importance of this teamwork approach is clearly evident. Furthermore, it is obvious that these developments were essential in keeping with the advances made in other medical specialties, particularly with reference to cardiovascular surgery. Many innovations in the concepts of anesthetic administration for all types of patients have arisen as a result of knowledge gained in experiences with intracardiac surgery, extracorporeal circulation and hypothermia. In dealing with the patients who require such procedures, the anesthesiologist has become increasingly aware of the deleterious effects of hypoxia and hypercarbia associated with hypoventilation and inadequate circulation to vital areas. Accordingly, the need was seen for practical and accurate devices with which the important physiologic parameters could be monitored simultaneously. By recruiting the aid of electronics engineers, such equipment has been made available and is rapidly becoming more practical and economical for general usage.

NONFLAMMABLE INHALATION ANESTHETIC AGENTS

As an outgrowth of the increasing utilization of electrical equipment in the operating suite, there has also been a renewed and necessary interest in the development of nonflammable inhalation anesthetics. The organic chemist has responded well to this necessity by intensive research with compounds of known anesthetic potency and by synthesis of previously unknown compounds which, at least chemically, would appear to satisfy anesthetic requirements.

Less than 4 years ago, Raventós[11] published the pharmacologic properties of a compound prepared by Suckling and which is now well known as halothane (Fluothane). The increasing popularity of halothane in clinical anesthesia attests to the fact that the drug represents a most important advance in the field of inhalation anesthesia.

Very early in the investigation of halothane, it was realized that the primary disadvantage of this compound was its great anesthetic potency which obviated its safe usage with conventional vaporization equipment. This fact led to the development of two vaporizers designed specifically for use with halothane[7, 8] and increased the importance of the "copper kettle" vaporizer described by Morris in 1952.[10] The development of the latter instrument has also proved to be of great value in the investigation of several newer fluorinated hydrocarbon compounds.[1, 6]

GAS CHROMATOGRAPHY

In assessing the useful concentrations of these new anesthetic compounds, and in aiding in the detection of abnormal levels of oxygen and carbon dioxide in anesthetic atmospheres and in blood, the quantitative analysis of such values has assumed considerable importance. Although instruments for this purpose have been available for many years in industry, their medical application has only recently been explored. Infrared analysis has been shown to be satisfactory for determination of halothane concentrations in inspired atmospheres[9] and has been used for many years in analysis of carbon dioxide. During the past year, we have been particularly impressed with gas chromatography as an all-purpose analytical device, in that all components of an anesthetic mixture may be analyzed quan-

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titatively in a few minutes and from a single sample.[5]

IMPROVED DESIGN OF ANESTHESIA MACHINES

In addition to the developments mentioned in the preceding paragraphs, safety during inhalation anesthesia has been increased considerably by improvements in the design of anesthetic machines and greater standardization of ancillary equipment. A notable example is that of the "pin-index" system which virtually eliminates error in attachment of cylinder gases to the anesthetic machine.

Enumeration of the many advances in design of mechanical ventilators and oxygen therapy equipment during the past few years would not be possible in an article of this nature, but the importance of such developments is notable.

LOCAL ANESTHESIA

In the field of local anesthesia a promising new drug, Mepivacaine (Carbocaine), has been introduced recently and in preliminary studies has proved equal to or better than lidocaine both in onset of action and duration of anesthesia. This drug has been used for virtually all types of conduction anesthesia during the past 6 months at this institution and in the authors' experience, Carbocaine has actually proved to be superior to lidocaine in the above respects. In addition, complaints of burning pain during injection have been few, in contradistinction to those associated with the use of lidocaine. Studies concerning the usefulness of Carbocaine as a topical anesthetic are planned for the immediate future.

PARENTERAL ANESTHETIC AGENT

The investigation of drugs which may be used as parenteral anesthetics has thus far failed to produce a compound superior to thiopental or thiamylal. There is little doubt, however, that continued interest in this phase of anesthesia will eventually result in the development of such a drug.

MISCELLANEOUS OTHER CONSIDERATIONS

In addition to the truly new developments in anesthesiology, there has been considerable research devoted to re-evaluation of drugs and techniques which have been a part of anesthesiology for many years. For example, since the advent of halothane anesthesia, a reassessment of chloroform anesthesia has been instituted by a few investigators.[2] In spite of the controversy which has arisen as a result of the continued use of this drug, it is important to re-evaluate such compounds critically under different conditions before completely shelving them for all time.

An interesting development also associated with the introduction of halothane has been its mixture with diethyl ether in an approximate ratio of 2:1 by volume. This mixture actually forms a new compound known as an azeotrope and which in this instance has been termed "fluether." Dobkin and others[3] have investigated the properties of this mixture and have reported the azeotrope to be safer than halothane alone because it induces less respiratory and cardiovascular depression. Confirmation of this work in certain other areas is yet to be established.

With the increasing expense of anesthetic gases and volatile compounds, the closed circuit method of anesthesia, wherein low flows of gases are utilized, definitely offers greater economy than does the partial rebreathing or nonrebreathing methods. A comparative re-evaluation of these techniques is under way in an attempt to determine which method is actually the safer for practical purposes. The results of this work will be presented in a subsequent publication.

POSTANESTHETIC COMPLICATIONS

The prevention of postanesthetic complications, and of those which have occurred insidiously during anesthesia but remain unnoticed until later, has received greater attention recently. For example, our evaluation of acute and chronic hypovolemic states is more critical, as is the careful questioning into the patient's history regarding his use of steroids, "tranquilizers" and antihypertensive drugs. By giving attention to these details, there is little doubt that many severe and perhaps catastrophic hypotensive episodes have been prevented or treated in time to avoid this complication.

The problem of unintentional hypothermia, especially in infants and small children, as a result of air conditioning, moist drapes and infusion of cold solutions is still another important facet of clinical anesthesia which has received too little attention.

Severe lowering of body temperature has been observed in infants undergoing thoracotomy, abdominal explorations and other surgical procedures. Recently we have observed a body temperature of 80°F. in an infant receiving exchange transfusion.

As a result of the observation of prolonged hypothermia and the shock-like status produced by cold in infants, the routine monitoring of body temperature during surgery has assumed great importance. Stephen[12] has recently reported experiences with a device which automatically maintains the temperature of the patient at any predetermined degree. This unit has proved valuable in the prevention of marked elevation or lowering of body temperature, and wider usage of such devices is anticipated.

In the future of anesthesiology, one can foresee the solution of many of our problems. It is not inconceivable that in our lifetime we may learn the fundamental principles of the production of narcosis which still remain obscure. As a result, we may also find the "ideal anesthetic."

SUMMARY

A few of the many advances in anesthesiology during the past few years have been presented briefly as representative of certain facets of the specialty in which progress has been made.

Emphasis has been placed on the fact that such progress has resulted directly from an increased cooperative effort among anesthesiologists, surgeons and other clinical specialists and expanding communication and consultation with basic scientists.

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SURGERY OF THE FACIAL SKELETON*

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The facial bones should be looked upon as the all important framework of the face. Delicately balanced muscles which produce expression, orbital function and assist in respiration, speech and mastication arise from this framework and insert in other areas of the framework, or attach to and move the over-all covering of the face, the skin. In some areas, subcutaneous tissue acts as a sort of cushion and allows the skin to move greater distances over the skeleton.

A change in the shape or position of the bones is visible as a change in the contour of the face. Also a change in the shape of the underlying skeleton throws the delicate balance of the muscles out of kilter so that the covering of skin is not held in the proper balance and movements of the facial muscles are not even.

In planning surgery on the facial bones, a correct diagnosis of the pathology is essential. Four fundamental and easily executed means of examination are available in order to establish a diagnosis.

EXAMINATION OF THE FACE

1. Look. When the patient is sitting, the examiner should sit directly in front of him, and when the patient is supine the examiner should, if possible, place himself at the head of the patient looking directly down onto the upside-down face. The face should then be looked at from all angles, so as to clarify the shape and to see shadows, prominences and depressions. Signs of trauma, inflammation, infection, changes in texture, color and ranges of motion should be noted.

2. Test. When the patient is able to cooperate, the examiner should test the visual field and should also test for hearing, smelling and sensation, at least on a gross level. He should also check movements of eyes, eyelids, lips, tongue, jaws and segmental groups of facial muscles.

3. Feel. Whether the patient is able to help or not, the examiner can obtain the most information by palpating both sides of the face at the same time with a gentle but firm pressure. One should feel for crepitation under the skin, bony irregularities, movements and tenderness.

An established routine of palpation is indicated. A good routine is: orbital margins, zygomatic arches, zygomata, nose, maxillae (externally as well as superior gingival sulcus), maxillary alveolus and teeth, mandible (externally and internally), mandibular alveolus and teeth, condylar region of the mandible.

4. X-ray. X-ray examinations are made last and are valuable when used in correlation with the other examinations but should never take precedence over the physical findings of an experienced examiner. Views for radiographic study are determined by the findings thus far in the examination.

The usual radiographic studies made on the facial bones are:

1. Skull: posterio-anterior and lateral for study of cranial vault proper and an over-all screening.

2. Modified Waters view of the face. This view gives the most information about the middle third of the face: orbital margins, zygomata and maxillae.

3. Mandible: posterio-anterior and laterals, exaggerated posterio-anterior. The latter gives valuable information about the condyles, condylar necks and ascending rami.

4. Towne's view of the skull. The zygomatic arches are usually outlined clearly.

5. Temporo-mandibular joint view in open and closed position. These may be helpful when used with the exaggerated posterio-anterior and lateral views.

SURGERY

After a thorough history and examination have been completed, the steps for correction must be planned for that particular case.

Some of the more common deformities will be

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taken up along with a brief resumé of treatment. Some specific cases will be illustrated.

SURGERY OF THE NOSE

The nose is a prominent and vulnerable feature of the face which is frequently misshapen after fracture. The nose is the feature of the face most commonly operated upon, and some of the most pleasing results can be obtained from surgery upon the nose.

The techniques for the corrective surgery of the nose are:

1. Nasal reconstruction with or without submucous resection or septal revision.
2. Implantation of bone or cartilage to raise the dorsum and tip of the nose.

Nasal operations are usually done under local anesthesia, but general anesthesia may be used. In the case of the prominent or hump nose, intranasal incisions are used. The bony and cartilaginous hump is removed with saw, osteotome or double actioned bone cutter. The nasal bones are cut at the cheek level and pushed in to narrow the dorsal line. The septum is then shortened to compensate for the lowering of the dorsal line. The alar cartilages are trimmed to narrow the tip to fit in with the over-all nose.

Frequently the nose with a large hump or crook has an obstruction to the airway produced by a badly deviated or buckled septum. In properly lining up the nose, this deformed septum is corrected by repositioning or submucous resection.

The nose without its supporting structure, which has been lost by disease or trauma, can be given a ridgepole of bone or cartilage.

SURGERY OF THE MANDIBLE

Prognathism. Correction of this condition is indicated for both cosmetic and functional improvement, since the teeth usually do not occlude.

Dental models for study and planning of the procedures are essential. When the best occlusal position has been found, the proper position for and proper type of osteotomy can be chosen. If, when the teeth are shifted, they occlude without change of the contour of the body of the mandible, a high ramisection above the lingula is good. On the other hand, when the body of the mandible must be changed in order to obtain occlusion of the teeth, a step osteotomy or ostectomy can be done through the body.

FIG. 1. Prognasthism with open bite (case 1)

FIG. 2. Profile after sliding osteotomy of mandible through ascending ramus (case 1).

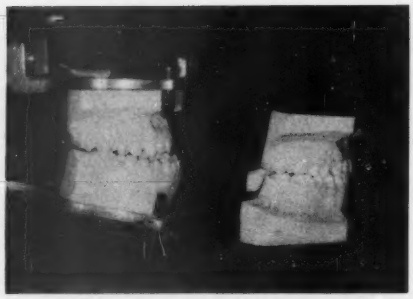

FIG. 3. Dental models with preoperatives on articulator (case 1).

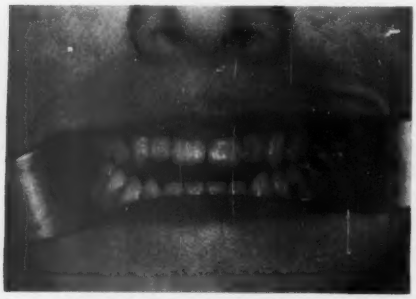

FIG. 4. Occlusion preoperatively (case 1)

A nasal endotracheal tube for anesthesia is essential.

For the high ramisection, a rather short incision (3.5 to 4.0 cm.) is made in front of the lobule of the ear and extended down toward the angle of the mandible. Once the skin has been penetrated, the ascending ramus is approached by blunt dissection, the muscle fibers are spread, the posterior border of the parotid gland is elevated, and the posterior border of the ascending ramus is found. The lateral aspect of the ramus is cleaned all the way to the anterior margin. The notch is located, and then it is best to drop down approximately 1 cm. for the osteotomy. A template of sterilized x-ray film, that has been prepared to show the angle of the cut from study of the dental models and the x-rays, is applied over the face and the proposed line of cut is marked on the skin. With the soft tissues gently retracted to expose a narrow strip across the ramus, the osteotomy is done. The cut can be made with a vibrating saw, or multiple drill holes can be made and the final fracture made complete with a small osteotome. A saw can also be used.

When both sides have been severed and the main portion of the mandible is free, one member of the team (usually the oral surgeon) puts rubber bands in place on the arch bars which have previously (usually the day before) been applied to the teeth. The fracture sites are inspected for apposition of the bone ends. If there is an excessive amount of bone protruding posteriorly, it is rongeured off and then the wound is closed in layers without drains. Every effort is made to obtain fine line scars.

The patient is usually in the hospital only 2 to 3 days, but the elastic traction is kept on the teeth for approximately 7 to 8 weeks.

FIG. 5. Teeth in occlusion while healing (case 1).

FIG. 6. Occlusion after healing (case 1)

FIG. 7. Retruding mandible secondary to infection in childhood with loss of lower teeth and ankylosis of temporomandibular joints (case 2).

FIG. 8. Front view (case 2)

FIG. 9. After sliding osteotomy through ascending ramus, moving mandible downward and forward, and dermagraft to temporomandibular joint regions (case 2).

ANKYLOSIS OF THE TEMPOROMANDIBULAR JOINT

The fused joint is approached through a pre-auricular incision and the bony ankylosis broken up, or a thin segment of bone immediately inferior to the condyle is resected. The contoured condyle in its newly created joint space is draped with a dermal graft (Georgiade, Altany and Pickrell). A regular diet is resumed almost immediately and gum chewing is prescribed. The essentials of the technique are similar to that just described for ramisection except for the dermal graft which is sutured or tied into position over the new head. Drill holes through the upper end of the ramus aid in securing of the graft.

Autogenous cartilage can also be used as an insert into the gap (Longacre).

SURGERY OF THE MAXILLA

The upper alveolus may be contracted secondary to a cleft palate deformity or to malunited fractures. Dental models are made first for study. Orthodontic appliances are made and the fixed portions put on before surgery. Fracture expansion of the alveolus and hard palate is done with a sharp thin osteotome and then the center section of the orthodontic appliance is applied to complete the surgery. No suturing of mucosa is required, but a pack may be applied. Thus, there is no need for interdental traction or fixation in some cases. The appliance maintains the desired

FIG. 10. Front view (case 2)

FIG. 11. Opening after Surgery, lower fall denture, and upper partial denture (case 2).

position of the alveolar fragments until healing has occurred, then a prosthesis with teeth to fill out the central section of teeth is made to be worn continually.

A cross-lip flap in indicated also in many of these patients in order to obtain the proper lip contour.

SURGERY OF THE ORBITAL MARGINS

Deformities of the orbital margins which are most amenable to surgical correction are those which are secondary to malunited and malaligned fractures. The problem is to restore the eyeball to its proper level and position in order to correct diplopia, to improve the cosmetic appearance of

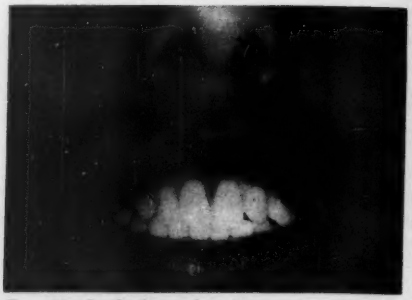

FIG. 12. Occlusion after Surgery as in Fig. 7, and dental prostheses as in Fig. 11 (case 2).

FIG 13. Old depressed fracture of floor of orbit, *right*, with diplopia and shrinkage of periorbital fat (case 3).

FIG. 14. After cartilage graft to floor of orbit and derma fat graft to upper lid (case 3).

the orbit and eye, and to round out the contour of the periorbital structures.

The means of accomplishing the desired changes are:

1. Onlay grafts of cartilage or bone to fill in the deficit and camouflage the deformity. The supraorbital rim can be filled in to round out the contour. The infraorbital margin and prominence of cheek can be rounded out, and the floor of the orbit can be raised to lift the eyeball to the proper level.

2. Dermafat grafts can be inserted into the lids to fill out the contour of the periorbital soft tissue.

3. Refracture along the old lines of fracture and repositioning of the bony margins or components in their proper positions and relationships. This is usually the most difficult but often the most rewarding procedure in that the results more nearly approximate the normal.

SURGERY OF THE ZYGOMA AND ZYGOMATIC ARCH

The zygoma and zygomatic arch have a deformity without actually involving the orbital rim. A depressed zygomatic arch may impinge the coronoid process of the mandible and prevent the mandible from closing. In general, the same techniques as presented above are applicable here.

SUMMARY

The examination of the face should follow an established routine: look, test, feel and x-ray.

Planning of the survery may entail consultation with allied specialities to assist with planning, surgery and postoperative care.

Some specific examples of surgery are presented in brief with photographs.

THE CASE FOR BILATERAL EXPLORATION OF INGUINAL CANALS IN PEDIATRIC PATIENTS PRESENTING UNILATERAL INGUINAL HERNIAS*

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Inguinal hernia is the most common surgical problem in infancy and childhood. It is a congenital abnormality and the presenting problem should be considered in a light somewhat different to that of the adult inguinal hernia associated with muscular weakness. Elective surgical repair is advisable as soon as the diagnosis is made on an otherwise normal infant or child. Because of the congenital background, the report of 12 to 20 per cent clinical finding of bilateral hernias before surgery, and the frequent return of infants or children who had had an inguinal hernia repair and later developed a hernia on the opposite side, much interest has been given during the past few years to the advisability of exploring both inguinal areas in those patients who present a single unilateral hernia.

Rothenberg and Barnett,[8] reported that 74 per cent of the 50 children under 12 years presenting with one hernia were found to have bilateral hernias (table 1). Mueller and Rader[6] studied 90 children who were less than 6 years old and found unsuspected hernias on the opposite side in 65 per cent. Clausen, Jake and Binkley,[4] studying 164 children, observed an incidence of bilateral hernias in 48 per cent, and emphasized that 73 per cent of 41 infants less than 6 months of age had contralateral hernias. Gilbert and Clatworthy[3] reported a study of 164 cases in which 60 per cent were found to have a patent processus vaginalis on the unsuspected side. Kiesewetter and Parenzan[4] reported a significant study of this problem in infants less than 2 years old. They found that in 100 patients presenting unilateral hernias, 60 per cent had a contralateral hernia. In a parallel study of 237 cases in which a unilateral hernia had been repaired in infancy, during the same period, 31 per cent of these children had required surgery for a contralateral hernia at a later date. McLaughlin and Coe,[5] reporting on "blind exploration" in 108 children, found that 58 per cent had bilateral hernias and 19 patients had returned for a second operation. During the period that these significant statistical studies were presented seeking to justify the procedure of routine bilateral exploration for hernias in infancy and children, only one report has appeared in the surgical literature which did not agree with this approach. Schidler[9] reviewed personal data on 57 children who were less than 11 years old and commented that only two had developed a hernia on the opposite side during the period of his observation. This study obviously ignored the strong congenital implication in this problem and the fact that asymptomatic small hernia or patent processus vaginalis may later become symptomatic during childhood or adulthood.

EMBRYOLOGY

During the middle trimester, the developing testis descends from its original position at the lower pole of the kidney on the posterior abdominal wall into the iliac fossa (fig. 1). An outpocketing, the processus vaginalis, of the peritoneum accompanies the testis from a point, later known as the internal inguinal ring, into the scrotum. The processus vaginalis parallels the vas deferens and the spermatic vessels and, together with coverings derived from the layers of the abdominal wall, comprises the spermatic cord. The gubernaculum testis is a homologue of the ovarian ligament which allegedly connects the lower pole of the testis to the scrotum. As it shortens, it literally draws the testis into the scrotum by the 9th month. Normally, the distal end of the processus vaginalis becomes detached from the proximal end or the funiculus and forms the tunica vaginalis, while the upper or abdominal end closes off at the internal inguinal ring and the connecting portion of the processus vaginalis is obliterated.[1]

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Presented in the Forum on Surgical Progress at the Annual Meeting of the Southeastern Surgical Congress, New Orleans, Louisiana, March 22, 1960.

TABLE 1

Reports of exploration of both inguinal canals in children who present only one clinical hernia

	Cases	Age	Contralateral Hernia
		yr.	%
Rothenberg and Barnett (1955)	50	0–12	74
Mueller and Rader (1956)	90	0–6	65
Clausen, Jake and Binkley (1958)	164	0–2+	48
Gilbert and Clatworthy (1959)	164	0–4+	60
Kiesewetter and Parenzan (1959)	100	0–2	60
McLaughlin and Coe (1960)	108	0–12	56
Martin (1960)	55	0–12	83.6

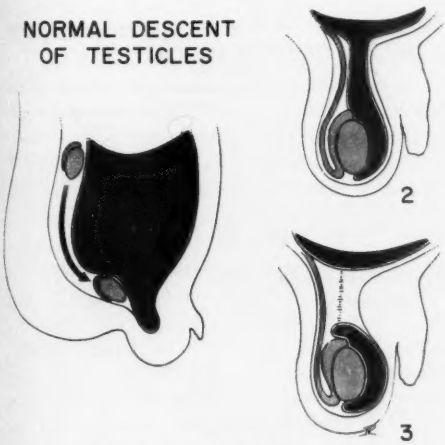

FIG. 1. Normal descent of testicles. (*1*) Migration from lower pole of kidney during middle trimester. (*2*) Processus vaginalis parallels the vas deferens and spermatic vessels into the scrotum. (*3*) Processus vaginalis normally becomes obliterated, leaving distal tunica vaginalis.

If this closure fails, a potential hernia exists (fig. 2). About 95 per cent of inguinal hernias are of the funicular type, resulting from simple failure of closure of the proximal processus vaginalis. In the remaining 5 per cent the entire processus remains open, producing a complete or scrotal hernia. Not infrequently, a narrow patent channel may exist between a scrotal hydrocele and the peritoneal cavity and is properly referred to as a communicating hydrocele. With cryptorchidism, the processus vaginalis remains open and associated hernia is commonly present.

STUDY OF CASES

Experience in applying this principle to the management of inguinal hernias in 75 consecutive children from age 1 month to 12 years during the period of January, 1958, through January, 1960, is reported (table 2).

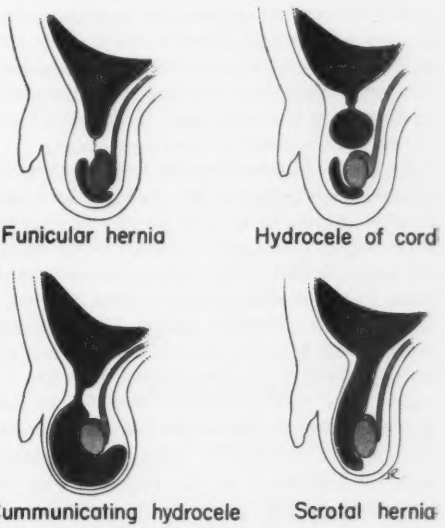

FIG. 2. Congenital inguinal hernias and hydroceles in children result from variations of failure of processus vaginalis to become obliterated.

TABLE 2

Analysis of 75 consecutive cases of children presenting inguinal hernias from January, 1958, to January, 1960

Procedure	No. of Cases
Unilateral inguinal hernia, bilateral exploration	55
Bilateral hernia, diagnosis before surgery	4
Previous repair, hernia on contralateral side	6
Unilateral hernia repair, opposite side not explored	10
Total	75

Fifty-five patients were admitted to surgery with a unilateral inguinal hernia and underwent exploration of the opposite side at the time of surgery, with the result that 83.6 per cent of these children were found to have an unsuspected hernia or a patent processus vaginalis. In all fairness to this study, the finding on exploration of the blind side was not considered positive unless the hernia sac or the patent processus vaginalis extended 2 cm. or more into the spermatic cord. In most cases with a patent processus vaginalis, the opening extended to or beyond the external inguinal ring. In all instances, the peritoneum was clearly identified at the level of the internal inguinal ring. This was believed necessary to establish with certainty whether or not an extention of the peritoneal process projected into the spermatic cord.

Four patients were observed to have bilateral hernias before surgery. Six patients had had unilateral hernia repair from 1 to 8 years before presenting with an inguinal hernia on the opposite side, theoretically further increasing the actual incidence of bilateral hernias in children.

During the period of study, 10 additional patients had unilateral hernia repair; however, the opposite side was not explored for a specific reason. Five children were treated for an undescended testicle, and at the beginning of the study we were reluctant to increase the operating time for exploration of the opposite side. As the procedure has become more routinely accepted, however, and because the operating time for such an exploration does not exceed 15 to 20 minutes, exploration is now performed as a matter of routine even in these cases. Five children, ages 4 through 9, have had bilateral exploration at the time of surgery for an undescended testicle and hernia on one side. Three of these, or 60 per cent, were found to have an unsuspected hernia on the opposite side.

It is believed that contralateral exploration is advisable only on elective patients whose general condition is excellent, on whom the anesthetic is proceeding satisfactorily, and on whom the original side has been expeditiously completed. The procedure is not advised for infants or children considered a substandard risk who may have had recent strangulation or incarceration with obstruction, or in whom the surgery for the original side had been tedious or difficult.[2]

The development of an inguinal hernia in a girl follows the same developmental pattern. The processus vaginalis descends alongside the round ligament and its coverings derived from the abdominal wall to the labium majora, occupying the canal of Nuck. A hydrocele of the cord, a hydrocele of the canal or an inguinal hernia may develop. Though inguinal hernias are more common in boys, one in 10 is observed in girls. In this group there were 10 girls from 2 months to 6 years, and nine of these, or 90 per cent, had bilateral hernias (table 3).

The right testis is thought to descend later than the left; this delay is believed responsible for the increased clinical incidence of right inguinal hernias. Thirty-five, or 64 per cent, of the unilateral hernias in this series occurred on the right side. Because of the high incidence of right inguinal hernias, pediatricians and surgeons have

TABLE 3

Incidence of contralateral hernia found on bilateral inguinal exploration according to location and sex

	No. of Cases	Contralateral Sac or Patent P.V.* at Operation	Percentage
Right herniorrhaphy; left exploration	35	28	80
Left herniorrhaphy; right exploration	20	18	90
Males	45	37	82
Females	10	9	90

* Processus vaginalis.

TABLE 4

Incidence of contralateral hernia found on bilateral exploration according to age

Age Group	No. of Cases	No. of Cases with Contralateral Hernia	Percentage with Contralateral Hernia
1 month–1 yr	13	13	100
1 year–3 yrs	12	12	75
3 years–6 yrs	20	15	75
6 years–8 yrs	4	3	75
8 years–12 yrs	6	6	100
Total	55	46	83.6

hypothecated that if the original hernia is on the left side, the statistical chance of finding an unsuspected hernia on the right is even greater. Swenson[10] stated that patients with an initial left hernia have approximately a 50 per cent better chance of developing one on the right side. Potts,[7] although not advising routine bilateral herniotomy, believes that a hernia on the left justifies advising bilateral operation. Clausen and coworkers,[2] Kiesewetter and Parenazan,[4] and McLaughlin and Coe[5] observed a high incidence of positive findings when the original hernia was on the left. In this series 90 per cent of 20 children with an original left inguinal hernia were found to have a hernia on the right, whereas 80 per cent of 35 children with an original hernia on the right were found to have a hernia on the left. This differential in such a small series is not statistically significant except to point out that regardless of the initial side, the incidence of an unsuspected hernia is sufficient to warrant bilateral exploration.

Opponents of the application of this procedure have made the observation that there is a higher incidence of bilateral hernias or patent processus vaginalis during the first 6 months to 1 year. Rothenberg and Barnett[8] found that 100 per cent of 12 cases had bilateral hernia. Clausen and associates[2] reported 73 per cent in infants less than 6 months of age. All 13 infants in this series less than 1 year of age were observed to have bilateral hernias. Zimmerman and Anson[11] assumed that a large percentage of these open processes close simultaneously during the first few months of life but rarely close spontaneously after 6 months of age. Review of these children by age groups reveal that there is a high incidence of bilateral hernia in each group and that all six of the children in the 8- to 12-year-old group were found to have a hernia on the opposite side (table 4).

Kiesewetter and Parenzan[4] observed that 65 per cent of the patients under 6 months and 56 per cent between 6 and 12 months were found to have hernias on the contralateral side. In the group of patients which they followed for the development of a second hernia, they concluded that the greatest danger of occurrence is in the earliest years after the occurrence of the first hernia. Their study arbitrarily shows 2 years as the upper limit. However, they indicated that the findings in older children might revise this limit upward. At the present time, it is believed that this practice should be followed in all children up to the age of puberty.

SUMMARY

1. The findings reported herein substantiate those of others that bilateral exploration of the inguinal canals in pediatric patients presenting unilateral inguinal hernias is justified and advisable.

2. In 83.6 per cent of 55 cases, an unsuspected hernia or patent processus vaginalis was found on the side on which it could not be demonstrated before surgery.

3. The extended operation requires 15 to 20 minutes and in the otherwise normal child adds very little risk because of anesthesia or surgery.

4. There is no prolongation of hospitalization or convalescence.

5. The need for a second hospitalization, anesthetic and operation is eliminated in a significant group of patients. It is quite possible that the vast majority of indirect inguinal hernias which require treatment in adults are in truth the end results of small, usually asymptomatic, clinically unrecognizable hernias which have been present since birth.

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SERUM AND TISSUE ELECTROLYTE CHANGES DURING PROFOUND HYPOTHERMIA*

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Many investigators have reported the electrolyte pattern alterations occurring in blood and tissue during hypothermia in the range now widely clinically applicable, *i.e.*, 20 to 30°C.[1, 5, 17, 19, 20, 22-24, 27]

The tremendous clinical potential offered by profound hypothermia (to 5°C.) necessitates investigation in this selective field to evaluate the attendant physiologic effects. It has been our purpose in these experiments to determine the ionic shifts which evolve during profound hypothermia.

MATERIALS AND METHODS

Large, healthy mongrel dogs, weighing from 17 to 25 kg., were anesthetized with pentobarbital sodium, 30 mg. per kg., and artificial respiration was maintained with room air through an endotracheal tube. Rectal, esophageal, blood and thigh muscle temperatures were constantly monitored by thermistors. Serial EKG recordings were made throughout the procedure, and mean arterial blood pressures were recorded by a mercury manometer attached to a femoral arterial catheter.

A midline sternal splitting incision was made from the suprasternal notch to just above the umbilicus, with meticulous hemostasis. Oozing at the thoraco-abdominal incision was controlled with thrombin-soaked gauze, and the incision was approximated with several large retention sutures in order to insure adequate internal cooling. Both femoral vessels and the right external jugular vein were exposed and catheterized after the dogs had received 50 mg. of heparin. Caval blood was withdrawn through the external jugular and the femoral vein catheters into a gravity venous reservoir and channeled into the pump oxygenator, while the cooled oxygenated blood was returned through a femoral artery. The cardiopulmonary bypass system consisted of a double roller pump, an Abbott plastic bag bubble oxygenator,† and a Brown-Harrison heat exchange unit [9, 10] which was cooled with iced water (fig. 1). Freshly drawn, matched whole blood was used to prime the extracorporeal unit, and circulation was maintained between 600 and 1200 ml. per minute, depending upon the rate of venous return which constantly diminished as the temperature fell.

The heartbeat consistently stopped between 19 and 15°C., and the respirator was discontinued at this point. When the esophageal temperature reached 5°C., the pump was stopped, the inflow and outflow portals clamped, and the dogs were left in complete circulatory arrest for 1 hour. No effort was made to prevent an upward temperature drift during this period. At the end of this time, the pump oxygenator was restarted, and the animal was warmed by the heat exchange unit with water at 40°C. As the heartbeat returned, artificial respiration was reapplied. The partial bypass warming system was maintained until the esophageal temperature reached 34°C., at which time the procedure was discontinued.

Thirty mg. per kg. of quinidine sulfate were given intravenously just before starting the partial bypass procedure, as advocated by Gollan.[18] On starting the rewarming phase, an additional 8 mg. per kg. of quinidine sulfate were given via the blood reservoir.

Arterial and venous blood samples, and heart, liver and rectus abdominis muscle biopsies were taken for analysis at the following intervals:

1. At normal body temperature before the bypass procedure began.
2. At 5°C.
3. After 1 hour of circulatory arrest.
4. Upon rewarming to 34°C.

Elliptical portions of left ventricular muscle were excised, and the defects were closed with interrupted mattress sutures of 3-0 silk. Wedges of liver tissue were removed, and gelfoam packs were inserted with interrupted sutures of 2-0 atraumatic chromic catgut. The rectus sheath

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† Kindly supplied by Abbott Laboratories.

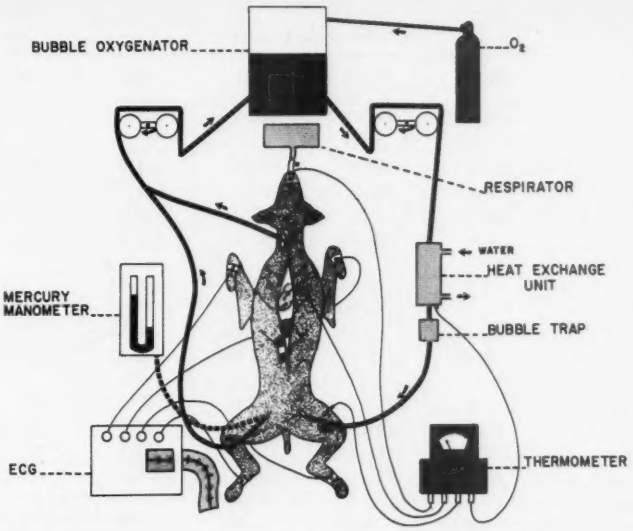

FIG. 1. Diagram of extracorporeal circulation in hypothermic dogs. Also shown are blood pressure, ECG, and temperature monitoring units.

was opened, and small slips of rectus muscle were likewise taken for analysis. All samples were large enough to render approximately 3 gm. of wet tissue for analysis. Immediately upon removal, the tissue was blotted with dry gauze and all excess connective tissue removed. The samples were quickly placed in ground glass, stoppered weighing bottles and stored at 4°C.

No attempt was made for survival with this group, since previous experiments here and elsewhere[18, 25, 26, 29] have demonstrated that prolonged circulatory arrest in deep hypothermia is compatible with life.

LABORATORY PROCEDURES

Determinations of the blood pH were made directly on a Beckman model G pH Meter. Serum sodium and potassium determinations were performed on the Beckman direct reading flame photometer. Serum chlorides were measured by the Eisenman[13] and Wilson and Ball[32] modifications of the Van Slyke method.[30] The determinations of calcium in serum were done by the method of Friedman and Rubin.[15]

Fat free, blood free tissue samples in the range of 2 gm. each were prepared for analysis by combination of the nitric acid digestion method of Flanagan, Davis and Overman,[14] and the procedure of Eichelberger and Miles.[12] After dilution of aliquots, sodium, potassium and chloride concentrations were determined photometrically as above. Tissue nitrogen content was determined by the Kjeldahl method of Meeker and Wagner.[21]

RESULTS

Results of the analyses are shown in table 1 and figures 2, 3, 4 and 5. For discussion purposes, the average values of the five dog series are taken.

Serum sodium and chloride increased during profound cooling and decreased toward the control value upon rewarming, with sodium going somewhat below the control level (fig. 2). Potassium followed the same trend but more dramatically, by increasing from a control value of 3.74 mEq. per L. to a level of 4.71 mEq. per L., and then falling to 2.79 mEq. per L. after rewarming. Inversely, calcium decreased from the control of 5.93 mEq. per L. to 3.74 mEq. per L. after 1 hour at 5°C. and then rose to 5.93 mEq. per L. upon rewarming to 34°C.

In both heart and skeletal muscle, sodium rose progressively, reaching its highest level after rewarming, whereas in the liver it increased during cooling but fell toward normal with rewarming (fig. 3). In inverse relationship to the serum, the chloride in heart, liver and muscle

TABLE 1

Serum electrolytes

	Dog 1	Dog 2	Dog 3	Dog 4	Dog 5	Average
	mEq./L.	*mEq./L.*	*mEq./L.*	*mEq./L.*	*mEq./L.*	*mEq./L.*
Sodium						
Control	138.7	138.9	140.0	144.0	133.0	133.9
5°C	138.1	137.8	140.0	144.1	133.0	138.7
1 hr. at 5°C	138.2	140.5	140.0	143.0	134.1	140.0
34°C	139.9	130.6	141.0	141.0	135.2	137.5
Potassium						
Control	5.22	3.12	3.40	3.81	3.21	3.74
5°C	4.77	3.72	3.90	3.32	3.38	3.82
1 hr. at 5°C	5.78	4.89	5.10	4.80	3.00	4.71
34°C	3.42	2.25	3.00	3.21	2.09	2.79
Chloride						
Control	82.0	110.4	98.8	105.4	106.8	100.7
5°C	93.2	104.8	109.4	101.6	108.4	103.7
1 hr. at 5°C	109.2	111.2	119.8	117.0	108.0	113.0
34°C	85.0	116.4	108.4	116.8	111.2	107.6
Calcium						
Control	4.48	3.60	6.36	7.07	8.13	5.93
5°C	4.94	4.64	6.56	3.24	4.52	4.80
1 hr. at 5°C	3.54	5.25	3.07	2.85	3.99	3.74
34°C	5.00	6.09	4.50	6.94	7.10	5.93

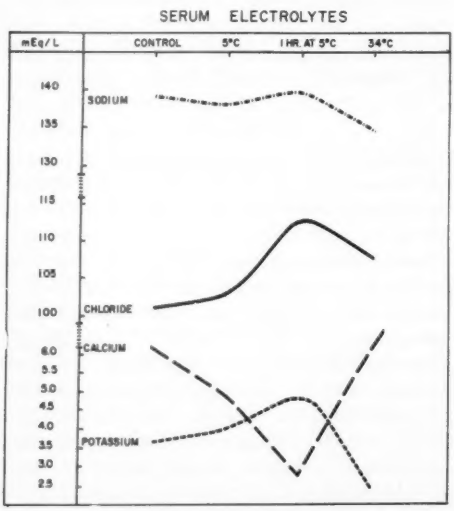

FIG. 2. Average changes seen in serum sodium, potassium and chloride during hypothermia to 5°C. with circulatory arrest for 1 hour.

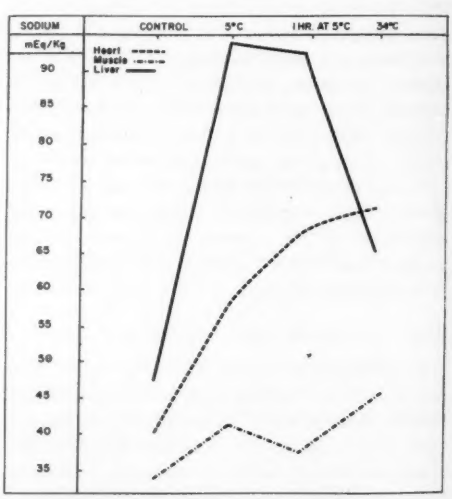

FIG. 3. This chart sho s the average changes in tissue sodium in cardiac muscle, skeletal muscle and liver occurring during hypothermia to 5°C. with circulatory arrest for 1 hour.

fell during cooling, but rose to a higher level after rewarming (fig. 5). Heart and muscle potassium increased with cooling and fell approximately to the control value during rewarming, whereas liver potassium markedly decreased on cooling and rose only slightly upon rewarming (fig. 4). During cooling, total nitrogen content fell slightly in skeletal muscle and markedly in the

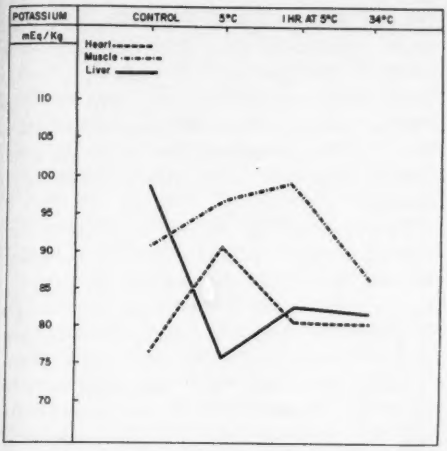

FIG. 4. This chart shows the average changes in tissue potassium in cardiac muscle, skeletal muscle and liver occurring during hypothermia to 5°C. with circulatory arrest for 1 hour.

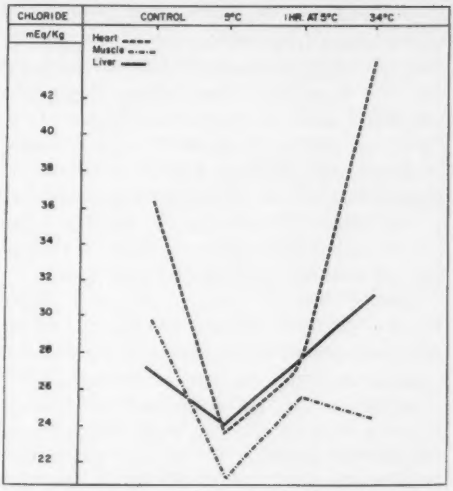

FIG. 5. This chart shows the average changes in tissue chloride in cardiac muscle, skeletal muscle and liver occurring during hypothermia to 5°C. with circulatory arrest for 1 hour.

liver, but both returned to normal upon rewarming.

In resumé, sodium was found to rise in all tissues, remaining high after rewarming, whereas in the serum, sodium rose only slightly and then fell to below control levels on rewarming. During cooling, potassium was found to increase in the serum, heart and skeletal muscle, while it decreased in the liver. On the other hand, chloride fell in all tissues, but increased in the serum during cooling.

The electrocardiographic tracings show the usual pattern seen in hypothermia as previously described by Tysinger, Grace and Gollan.[29]

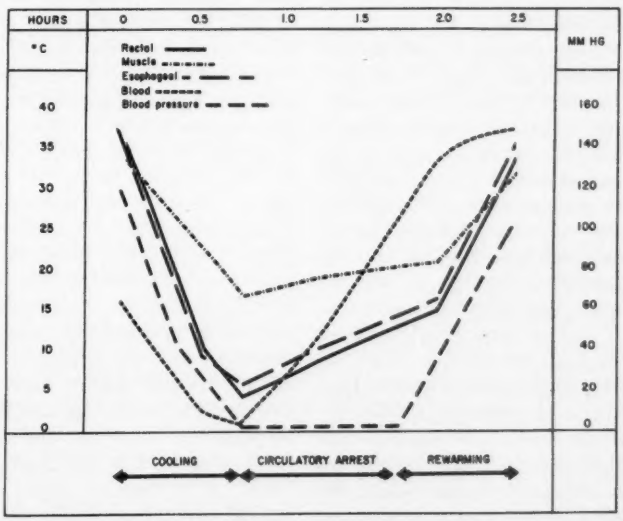

FIG. 6. Shown here is the differential in temperature of various areas of the body during profound cooling. Also illustrated is the mean systolic blood pressure during partial bypass cooling procedure.

This included a decrease in amplitude of P waves, decreased amplitude and prolongation of T waves, and prolongation of the QRS complexes and ST segments. These changes gradually progressed until the temperature reached 15 to 19°C., at which all electrical activity ceased. As rewarming took place, these changes reversed themselves with an essentially normal reading present at 34°C. Fibrillation was not seen in this series on cooling, but occurred twice with rewarming and was easily controlled by electric shock.

Control systolic blood pressures ranged between 120 and 140 mm. Hg (fig. 6). As the dogs were placed on the extracorporeal unit and cooling was begun, the systolic pressures fell to about 90 mm. Hg. The pressures drifted down to between 40 to 60 mm. Hg until the esophageal temperature reached 13°C., from which time they remained between 20 and 40 mm. Hg until 5°C. Upon rewarming, the pressures remained around 30 mm. Hg until the esophageal temperature reached 22°C. From this point the pressures ranged between 60 and 70 mm. Hg until 30°C., when the pressures reached 100 mm. Hg.

DISCUSSION

It has been shown by Munday[23] that in rats and rabbits under mild hypothermia (20 to 25°C.), serum sodium rises slightly, whereas potassium shows a marked decrease. Moulder[22] likewise reported that in mildy hypothermic dogs, plasma potassium tends to fall. Spurr and Barlow[27] found similar changes in sodium and potassium levels. Beavers and Rogers[5] found that in dogs cooled to 20° C., plasma potassium decreased, but that sodium and chloride showed little change. Our results during profound cooling show a slight hypernatremia and a more marked hyperkalemia. In analyzing the differences, it was of interest to note that during cooling to 5°C., the potassium rich liver tends to lose this ion in large quantities. Because of regional circulatory and physiologic differences previously noted by Brauer and his associates,[8] the liver cools more slowly than the blood or heart, as during hypothermia the decrease in hepatic blood flow is much greater proportionately than the fall in cardiac output. The fall in serum potassium observed in mild hypothermia may precede the later rise which we observed, because of the efflux of potassium from the liver as its temperature ultimately falls. In addition, the use of internal cooling with a pump oxygenator and blood transfusions, even though the latter were drawn on the same day, will certainly introduce many extraneous factors not present in simple external cooling. These differences may explain the discrepancy between the trend of serum potassium during mild and profound hypothermia.

Hypothermic dogs without adequate ventilation (artificial respiration) develop respiratory acidosis and would be expected to exhibit a moderate to severe hyperkalemia. Bigelow and his associates[6] observed a high potassium content in hypothermic dogs which were cooled without artificial respiration and were thus acidotic. On the other hand, Axelrod and Bass[1] found a fall in serum potassium in hyperventilated alkalotic dogs. Boere[7] found the same fall in potassium in well ventilated hypothermic patients. Thus, the relationship between cold, respiratory acidosis and serum potassium content is clear. Such a respiratory acidosis with a resultant hyperkalemia was prevented in our experiments by full ventilation so long as the heart was beating. Additional studies in our laboratory have the confirmed that under these circumstances of profound cooling, the blood pH, CO_2 and O_2 saturations are maintained within normal limits.[31] With well ventilated dogs, one would anticipate the hypokalemia associated with respiratory alkalosis, as mentioned by Munday.[23] By this reasoning it is felt that the hyperkalemia observed during profound cooling is a real phenomenon and not just a reflection of acid-base balance associated with CO_2 retention or a decrease in pH.

During cooling to 23°C., Gollan[16] reported a loss of potassium in the auricle. By contrast, Swan[28] observed an increase in myocardial potassium in dogs cooled to 30°C. Beavers and Rogers[5] believed that these differences in similar procedures were due to pH changes. Gollan's procedure involved hyperventilation with an increase in pH, whereas Swan's dogs were not ventilated and a respiratory acidosis resulted. In Beavers and Rogers' study, cardiac muscle exhibited an increase in potassium even though artificial respiration was employed in an attempt to maintain a stable pH. Spurr and Barlow[27] also found an increase in cardiac potassium under similar circumstances. Likewise, we found an increase in myocardial potassium during profound

cooling in which controlled respiration and presumably adequate perfusion were used. This fact, together with the increase in serum potassium observed, is encouraging, in that many workers feel that high cardiac and/or serum potassium content may be a factor in decreasing cardiac irritability.[2, 3, 5, 28] Beavers and Covino,[3] for example, obtained very striking results in reducing the incidence of fibrillation in hypothermic dogs by the administration of glycine. They felt that this might be explained by the attendant increase in serum potassium.

The mechanism by which the sodium increases in skeletal muscle, cardiac muscle, and liver during cooling and rewarming is not readily evident. Although serum sodium falls somewhat during this period, this fall is not quantitatively responsible for the changes which occur in the tissues. Spurr and Barlow,[27] as well as Beavers and Rogers,[5] reported slight decreases in tissue sodium associated with a concomitant rise in plasma sodium. Since profound hypothermia is fully compatible with permanent and relatively uncomplicated survival, it does not seem likely that physical disruption of the cellular membrane occurs which would allow an influx of sodium into the cell. It is possible that a temporary physiologic disruption may occur in hypothermia which causes slowing of the sodium pump mechanism. Although this may explain the changes during low temperatures, it would not explain the high sodium after rewarming. No studies have been done to determine how long sodium in the heart may remain high even after rewarming, but survival after this degree of hypothermia indicates that this is not irreversible.

The decrease in chloride was consistent in all tissues. In most areas of metabolism, sodium and chloride tend to follow each other, but such was not the case here. It is evident that the diffusions of sodium and chloride differ in muscle, in which sodium is such a mobile ion in playing its role in the processes of contraction. Here again the intrinsic properties of the cellular membrane during profound cooling may change to allow the exit of chloride from the cell. As expected, an increase in plasma chloride was found. Once again, if one accepts survival studies, the changes must be of relatively minor consequence.

Covino and Hegnauer,[11] by studying arterial and coronary sinus electrolyte content, found a loss in potassium and a gain in calcium in the hearts of dogs cooled to 23°C. Subsequently, Beavers and Covino[4] found that the administration of potassium chloride and ethylenediaminetetraacetate in dogs cooled to 25°C. deterred the deleterious effects (fibrillation) of a low serum potassium and the increased uptake of calcium by the heart. In dogs cooled to 5°C., we found an increase in serum potassium and a decrease in calcium which may be of some value in this regard. Nonetheless, the administration of massive doses of quinidine as suggested by Gollan[18] has proved essential in reducing the instance of fibrillation which otherwise is frequent with cooling, and almost routine in rewarming.

SUMMARY

Electrolyte changes have been determined by serial biopsies of the heart, liver and striated muscle and correlated with serum levels during profound hypothermia to 5°C. produced by partial cardiopulmonary bypass with a heat exchanger.

In contrast to the changes noted in mild hypothermia, potassium increases in the serum and striated muscle, whereas the liver reflects tremendous losses. Serum calcium shows a reciprocal change.

Sodium showed a much less dramatic increase both in the serum and biopsied tissues. Chloride rose in the serum while diminishing in the tissues during cooling, but returned toward normal on rewarming.

None of the observed changes appear to be of such magnitude as to disrupt function.

Acknowledgment. We acknowledge with gratitude the technical assistance of Mrs. Virginia Keith and Mrs. Lois Sampson in the biopsy analyses. This work was supported in part by grants from the National Institutes of Health (H-2806-C3) and the Mississippi Heart Association.

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DIAGNOSIS AND TREATMENT OF MALIGNANT SURFACE TUMORS*

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It is difficult to understand why patients and physicians alike are so apathetic about carcinoma that occurs on the surface of the body and yet become so concerned about carcinoma of the internal organs, because this is essentially the same process occurring in different locations. Squamous cell carcinoma of the skin will metastasize to regional nodes and eventually cause the death of the patient just as surely as bronchogenic carcinoma.

Of course, surface tumors have the unique characteristic of being seen in their earliest stages, and so can be diagnosed and treated early. In spite of this fact, however, the 5-year survival rate of resectable adeno-acanthoma of the skin is almost identical to the miserable 5-year results in bronchogenic carcinoma. Surface tumors, because of their early recognition and accessibility, should be 100 per cent curable, yet over 4000 people die in the United States each year from carcinoma of the skin alone.[4] Skin cancer is by far the most frequent malignant lesion, and its lethal potential must be appreciated if we are to increase survival rates.

The etiology of surface cancer is as nebulous as that of internal cancer, but we do know of certain predisposing factors. In the South, skin cancer is 2 to 3 times as frequent as in the northern states, probably because of longer and more intensive exposure to the rays of the sun.[11] Blond, fair-skinned individuals are much more susceptible than brunettes. That pigment is a protective factor can be shown by our own Tumor Board statistics. With an admission rate of roughly 2:1, whites to Negroes, the surface tumor percentage is 39:2.8. The ratio of surface tumors on exposed and unexposed areas of the white is 7:1, whereas in the Negro it is 1:1, demonstrating that pigmentation of the skin is as effective as clothing.[2]

Biopsy, of course, should be done on all questionable lesions, followed by adequate treatment. Large lesions may be partially excised with an area of adjacent normal tissue for biopsy (fig. 1), and small lesions may be completely removed with adequate margins as an excisional biopsy (fig. 2). Surface tumors should never be diagnosed by clinical characteristics alone. Exact histologic diagnosis is essential for adequate treatment. Adequate treatment for malignant lesions should be limited to either surgical excision or radiation. Fulguration may be used for premalignant lesions such as leukoplakia or senile keratosis only after histologic confirmation.

A three dimensional concept must be kept in mind in excising surface tumors. The most common mistake is to obtain an adequate peripheral margin and leave cancer in the base, simply because the depth of invasion is not appreciated (fig. 3). The majority of surface tumors can best be treated by surgical excision, primarily because serial sections can be taken of the periphery and base of the lesion and complete removal confirmed by the pathologist. This, of course, cannot be done with radiation therapy. Surgery also obviates the danger of radiation damage to surrounding tissue, including osteoradionecrosis and chondroradionecrosis.

One of the most commom problems seen in tumor clinics is the so-called farmers' or sailors' skin. The skin of these individuals has been preconditioned to malignant degeneration by many years of exposure to the sun and wind. The exposed areas are covered with senile keratoses (fig. 4). These people are instructed to protect their skin with lanolin and wear gloves and wide-brimmed hats when out of doors. They are recalled at 3-month intervals, and malignant lesions are excised.

The most frequent malignant surface tumors are the epitheliomas, with basal cell carcinoma occurring twice as frequently as the more malignant squamous cell carcinoma. Sex distribution is about 2:1, male to female, with the greatest age incidence after 40, although squamous cell carcinoma is not rare in the second and third decades.[6] Both lesions occur primarily on the

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FIG. 1. Incisional biopsy of large lesion, obtaining representative section of tumor and of adjacent normal tissue.

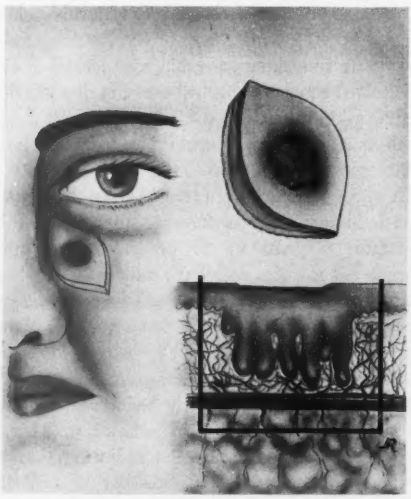

FIG. 2. Excisional biopsy of small lesion. Complete excision of tumor with adequate margins.

exposed areas of the body or in areas of chronic irritation. Squamous cell carcinoma has a predilection for the lower half of the face and neck, including the lips, cheeks, ears and dorsum of the hands; whereas basal cell carcinoma favors the nose, perinasal area, eyelids, temples and forehead, although it may occur in any of these locations, and diagnosis cannot be made by site alone.

SQUAMOUS CELL CARCINOMA (PRICKLE CELL CARCINOMA, EPIDERMOID CARCINOMA)

These may be ulcerated, nodular, scaly or indurated lesions originating from the prickle cell layer of the epidermis. They may resemble the senile keratoses from which they are often derived as flat spreading firm patches which ulcerate late in development and fade off into the surrounding skin. A papillary form also occurs

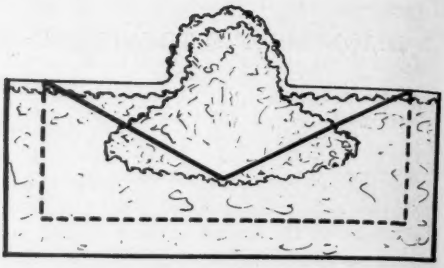

FIG. 3. Solid *V line* shows incomplete removal of lesion, leaving tumor tissue peripherally and in base. *Dotted line* demonstrates correct excision.

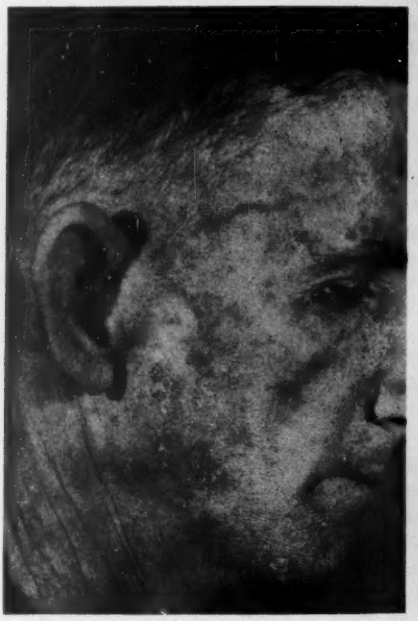

FIG. 4. "Farmers' skin" with multiple senile keratoses.

which may be pedunculated or sessile. Growth is usually much more rapid than basal cell carcinoma and ulceration occurs earlier. Squamous cell carcinoma cannot be definitely differentiated clinically from other skin lesions; consequently, biopsy is imperative for intelligent treatment. Cutaneous blastomycosis may closely simulate squamous cell carcinoma and, of course, the therapy is entirely different (fig. 5). Early diagnosis is extremely important, since squamous cell carcinoma is capable of producing not only

FIG. 5. Cutaneous blastomycosis. Similar lesions had been removed previously with the clinical diagnosis of squamous cell carcinoma.

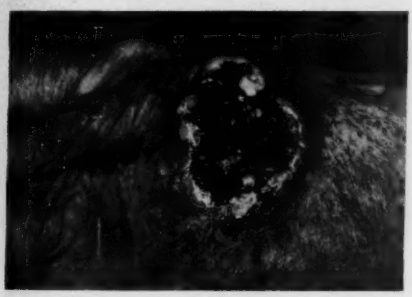

FIG. 6. Squamous cell carcinoma on dorsum of the hand. Lesion had been present for 5 months.

regional spread to nodes, but also distant metastases. Lesions of the trunk and extremities (fig. 6) metastasize earlier and more frequently than those of the face and neck. Squamous cell carcinoma may develop in areas of lupus erythematosus (fig. 7), radiation dermatitis (fig. 8), ulcerated burn scars (Marjolin's ulcer), chronic draining sinuses (fig. 9) and fistulous tracts (fig. 10). Although metastasis occurs later than in the usual squamous cell carcinoma, these lesions may be extremely difficult to eradicate

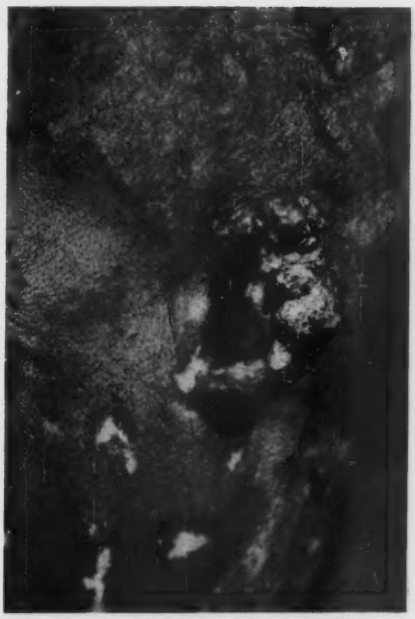

FIG. 7. Squamous cell carcinoma of ear in chronic lupus erythematosus.

FIG. 8. Squamous cell carcinoma occurring in area x-radiated for "skin cancer" 5 years previously.

FIG. 9. Squamous cell carcinoma in old chronically draining pilonidal sinus.

FIG. 10. Squamous cell carcinoma in fistula-in-ano. This 28-year-old man had had multiple fistulectomies in the previous 10-year period.

because of extensive scarring and involvement of adjacent tissue. Treatment of squamous cell carcinoma consists of wide local excision of the lesion with histologic verification of complete removal. Regional lymph node involvement occurs in only 5 to 20 per cent, depending on location; consequently, prophylactic dissection is not practical. However, if regional nodes are involved and the primary lesion is controlled, radical dissection should be performed. Carcinoma of the lip with extensive leukoplakia should be treated by wedge excision of the carcinoma and removal of the mucous membrane of the lip (lip shave) (figs. 11 and 12). Carcinoma of the upper lip is relatively rare and has much poorer prognosis than that of the lower lip (fig. 13).

BASAL CELL CARCINOMA (ADNEXAL CARCINOMA)

Clinically, this tumor exhibits a multiplicity of forms described by such terms as nodular (fig. 14), pigmented, ulcerated (fig. 15), Pagetoid, fungoid, carcinoid, morphea-like (fig. 16) and superficial (fig. 17), which simply re-emphasizes the fact that surface tumors can be accurately

FIG. 11. Squamous cell carcinoma of lower lip with extensive leukoplakia.

FIG. 12. The patient of figure 11 after V-excision of squamous cell carcinoma and "lip shave."

FIG. 13. Squamous cell carcinoma of upper lip

FIG. 15. Basal cell carcinoma of nasolabial fold, ulcerative type (rodent ulcer).

FIG. 14. Basal cell carcinoma of forehead, nodular type.

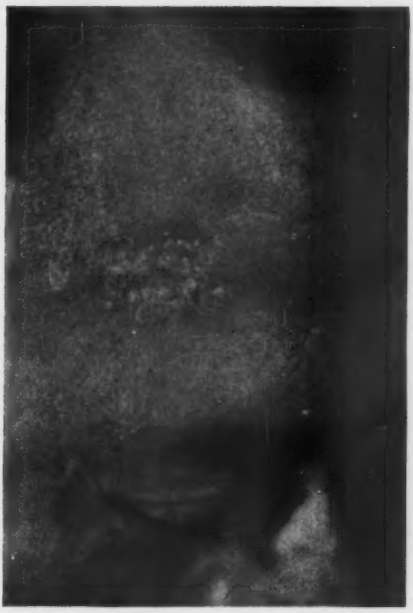

FIG. 16. Basal cell carcinoma of forehead, morphea-like type.

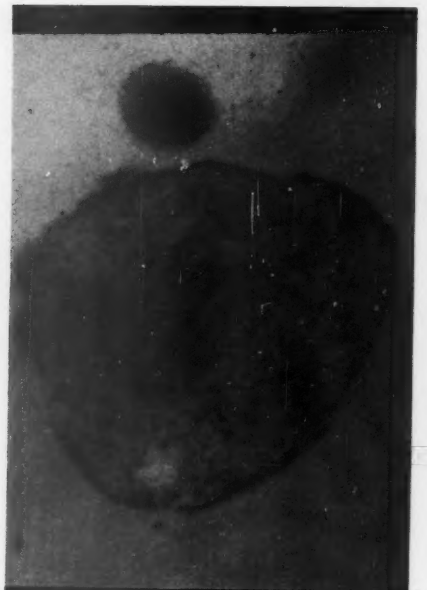

FIG. 17. Basal cell carcinoma of chest, superficial type, following "matchbox dermatitis."

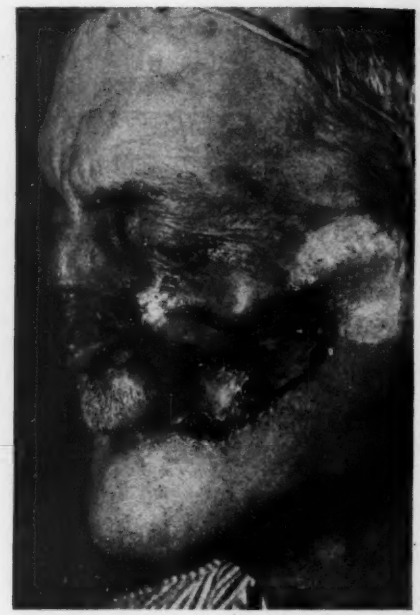

FIG. 18. Adeno-acanthoma of nose and cheek. Tumor is still spreading after multiple "complete excisions" over a 2-year period.

diagnosed only histologically. These lesions invade by direct extension and, if neglected or inadequately treated, may destroy vital structures, causing extensive deformity and even death. Metastasis to regional nodes has been reported in the literature, but is extremely rare.[9] For all practical purposes, these lesions can be considered as nonmetastasizing. The preferred treatment for basal cell carcinoma is complete surgical excision. X-ray therapy may be used for small (less than 2 cm. in diameter) lesions with good results.

ADENO-ACANTHOMA

Lever[10] originally regarded these tumors as sweat gland carcinomas composed of glandular and epidermal elements and explained their presence by the fact that sweat ducts are composed of squamous cells in their epidermal portion and glandular cells in the dermis. He now feels that it is possible that these tumors are squamous cell carcinomas of alveolar growth with individual cell keratinization resulting in acantholysis in the center of the alveolar formation. Clinically these tumors resemble squamous cell carcinoma and have a predilection for the head and neck.

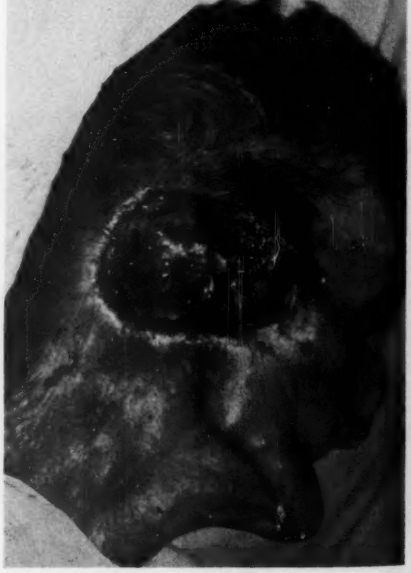

FIG. 19. Adeno-acanthoma of right postauricular area, terminal, from direct extension of tumor through mastoid to cerebrum.

They show shallow central ulceration and may have a verrucous surface. They spread much more rapidly by direct extension than the usual squamous cell carcinoma, eroding underlying bone and involving the cerebrum.[12] They will metastasize to regional nodes; however, in our patients, death has been due to direct extension to the brain rather than to metastases (figs. 18 and 19). This lesion has been the most lethal in our series. Four out of six patients have died in less than 5 years. Treatment consists of ultra-wide and deep excision to eliminate all involved tissue. Patients require frequent meticulous follow-up examinations.

MALIGNANT MELANOMA

The histogenesis of malignant melanoma is controversial. However, most pathologists agree that the typical melanoblast from which the melanoma develops originates at the dermo-epidermal junction and is of neuroectodermal origin. A presumptive diagnosis of malignant melanoma may be made when any nevus changes in pigmentation, size or elevation, or bleeds or ulcerates. A junctional nevus, because it is a known precursor of malignant melanoma, is particularly suspect (fig. 20). Malignant melanoma may develop on any part of the body, but is more common on the head, neck and lower extremities (figs. 21 and 22). They may occur at any age, but are not considered malignant before the age of puberty. Melanoma metastasizes more rapidly and more frequently than any other neoplasm. Tumor emboli may spread through the blood stream or the lymphatics. All nevi on the genitalia, palms of the hands or soles of the feet should be removed prophylactically, because they are almost always junctional.[3] Similarly, all nevi in areas of chronic irritation should be excised. Incisional biopsy of a suspected melanoma is condemned because of the danger of hematogenous spread. Also, local anesthesia should not be used because of the hazard of spread from needle trauma. Treatment is entirely surgical, because malignant melanoma is as radio-resistant as the skin itself and can only be affected by cauterizing doses of radiation.[7] The administration of chemotherapeutic agents systemically and by local perfusion techniques has not yet proved curative and at present is used as an adjuvant to surgery or for palliation.[5, 15]

FIG. 20. Junctional nevus in beard area of left cheek.

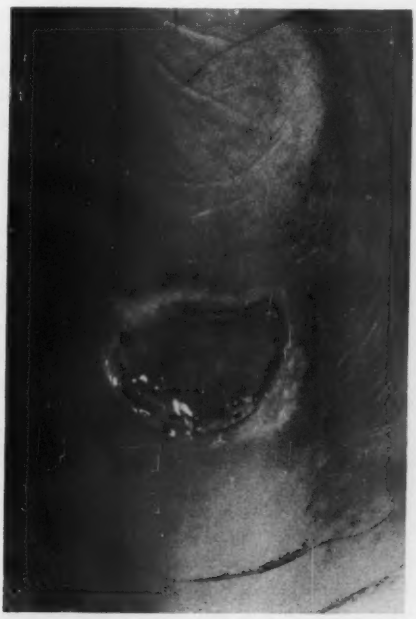

FIG. 21. Malignant melanoma, amelanotic type, on plantar surface of heel. Presented as an unhealed ulcer of 8 months' duration.

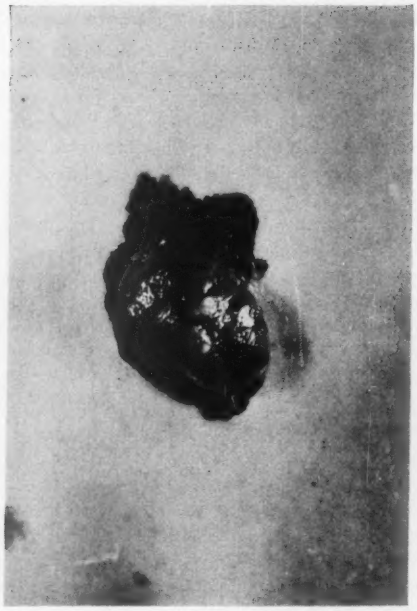

FIG. 22. Malignant melanoma on thorax arising from a pre-existing nevus.

Surgical excision should be wide, at least 10 cm. from the periphery of the lesion, and extend through subcutaneous tissue and fascia to underlying muscle. Regional node dissection should be done concomitantly and in continuity when practical.[14]

SARCOMA

A wide variety of primary sarcomas appear on the body surface. These tumors are of mesodermal origin and may occur in any location. They vary greatly in malignancy and rapidity of progression. Metastasis is more common by the blood stream than the lymphatics, and local recurrence following treatment is frequent because of wide local invasion.

Fibrosarcoma involving the skin and subcutaneous tissue usually presents as a firm circumscribed nodule which grows slowly and invades adjacent structures (fig. 23). They are relatively low grade malignant lesions that metastasize late in their course. Treatment of these radioresistant tumors consists of wide surgical removal.

Angiosarcoma is a malignant tumor of angioblastic origin. Clinically these are soft, bluish red lesions composed almost entirely of vascular sinuses. They may arise from pre-existing hemangiomas. Because of the extreme vascularity of the large tumors, x-ray therapy alone or combined with surgery is the treatment of choice.[13]

Rhabdomyosarcoma arises from striated muscle and may occur anywhere on the body surface. It presents as a firm, smooth painless tumor that cannot be differentiated clinically from other subcutaneous lesions. These tumors are highly malignant; treatment is surgical excision.

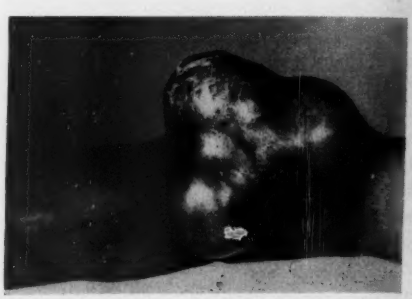

FIG. 23. Fibrosarcoma of wrist of 2 years' duration.

FIG. 24. Neurosarcoma of plantar surface of heel arising from a neurilemoma.

FIG. 25. Myxosarcoma of wrist, recurrent 6 months after excision.

FIG. 26. Liposarcoma of left scapular area, despite wide removal, metastasized to left cervical lymph nodes.

FIG. 27. Dermatofibrosarcoma protuberans. Early nodule formation.

Leiomyosarcoma arises from smooth muscle and may occur on any anatomical area. It can originate primarily from skin structures such as the smooth muscle of blood vessels or arectores pilorum. Diagnosis is made only by biopsy. Treatment is wide surgical removal.

Neurosarcoma (*neurogenic sarcoma*), a highly malignant tumor of peripheral nerve tissue, usually represents the malignant degeneration of neurilemmoma (fig. 24) or neurofibroma. Tumors of von Recklinghausen's disease may develop into neurosarcomas. Treatment is complete surgical removal.

Myxosarcoma is a cutaneous and subcutaneous tumor of low grade malignancy, characterized by multiple semicystic coalescing nodules (fig. 25). It may occur anywhere on the body, but presents most frequently on the extremities. Myxosarcoma usually invades by direct extension. Consequently, surgical excision should be complete with adequate margins, because recurrence is common.

Liposarcoma is an extremely rare malignant tumor which may occur in any area where fat is present (fig. 26). This tumor usually does not arise from a pre-existing lipoma, but bears a

FIG. 28. Dermatofibrosarcoma protuberans. Late, nodules coalesced to form single violaceous tumor.

TABLE 1

Composite 5-year survival rates for malignant surface tumors

Tumor	Percentage of Survival
	%
Malignant melanoma	21
With metastasis to regional nodes	14
Without metastasis to regional nodes	40
Adeno-acanthoma	25
Squamous cell carcinoma of skin and lower lip	80
Squamous cell carcinoma of upper lip	46
Basal cell carcinoma	93
Angiosarcoma	27
Leiomyosarcoma	55
Neurosarcoma	60
Rabdomyosarcoma	35
Fibrosarcoma	55
Dermatofibrosarcoma	69
Myxosarcoma	92
Liposarcoma	35

close resemblance to lipoma grossly. Liposarcoma may occur as multiple primary tumors or have satellite nodules around the main mass. They are not responsive to x-radiation and will metastasize to regional nodes. Treatment is wide surgical excision.

Dermatofibrosarcoma protuberans is a low grade malignant tumor which usually starts as firm cutaneous or subcutaneous nodules covered with normal epidermis (fig. 27). As growth progresses, these nodules may coalesce to form a large violaceous tumor mass (fig. 28). Growth is extremely slow; however, this tumor may metastasize. They occur most commonly on the trunk, but may present in any location. Dermatofibrosarcoma is radio-resistant. Surgical excision is the treatment of choice.

PROGNOSIS

An accurate prognosis for malignant surface tumors should take into consideration such factors as size and location of the tumor and histologic grading. Since this information is not available in reported series, a composite figure is presented for each tumor which includes all lesions in that group unless specifically noted (table 1 and references[1, 3, 4, 6-8, 10, 12-16]). This at best gives only an impression of the relative malignancy of these lesions.

In general, the prognosis of patients with malignant surface tumors depends on early recognition and adequate treatment of the lesion. Ideally, all these tumors should have a good prognosis. However, if they are not recognized in their early stages or if they are treated inadequately, the prognosis is poor.

CONCLUSION

Although we are constantly searching for newer and more exotic methods of discovering cancer earlier, we should not lose sight of the fact that in locations where cancer can be seen in its earliest stages, that is, on the surface of the body, we are still far short of our goal of 100 per cent curability.

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THE SURGICAL MANAGEMENT OF SALIVARY GLAND TUMORS*

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In the past decade, the surgical management of salivary gland tumors has become better understood. The results of treatment in various series of cases have shown improvement in cure rates with fewer recurrences.[1, 11]

The relatively high incidence of carcinomas in the salivary gland tumors removed in this community stimulated our interest in reviewing this group of cases.

MATERIALS

The charts and representative pathologic material were reviewed from those patients having salivary gland tumors removed at the University Hospital from its opening on July 1, 1955, to July 1, 1959. In addition, the records of all patients having surgical treatment of salivary gland tumors at the Mississippi Baptist Hospital and the Jackson Memorial St. Dominic's Hospital from January 1, 1954, to July 1, 1959, were reviewed. Twelve different surgeons and several resident surgeons at the University Hospital were the operating surgeons.

RESULTS

A total of 87 salivary tumors were removed (table 1). There were 73 salivary tumors of the parotid gland, seven of the submaxillary gland, one of the sublingual gland, and six salivary tumors arose from the minor salivary glands within the oral cavity and the hypopharynx.

There were 45 patients with mixed tumors of the parotid gland (table 2). Thirty-eight of these patients were operated upon primarily, and seven patients had recurrences of mixed tumors operated upon two or more times. Four patients had Warthin's tumors; one patient had a hemangioma; one patient had a hemangiosarcoma of the parotid gland; and there were 22 patients with carcinomas of the parotid gland.

Three patients were explored with a preoperative diagnosis of a parotid salivary tumor, but they were found to have sialadenitis. One patient was found to have a hyperplastic parotid lymph node. These latter four cases were not considered further, since these patients did not have salivary neoplasms.

The average age of the patients with mixed tumors of the parotid who were operated upon primarily was 39 years (table 3). The average duration of symptoms in this group was 2.7 years from the date at which the tumor was first noted to the time of its removal.

The average age of the 22 patients with parotid carcinomas was 56 years (table 3). The average duration of symptoms in this group of patients was 3 years. Two of these cases were of particular interest.

The first case, a 63-year-old white woman, had roentgen ray therapy of unknown dosage to a parotid neoplasm without prior biopsy. Ten years later, exploration of her parotid tumor was performed because of increasing size of the tumor, and the patient was found to have an adenocarcinoma of the parotid. Superficial parotidectomy 3 years ago has been followed by no recurrence thus far.

The second patient, a 27-year-old white male, had a left superficial parotidectomy for an acinic cell carcinoma. Two years later, a right superficial parotidectomy was performed for a mixed tumor. Bilateral parotid neoplasms are rare,[5] and this patient was the only example seen in this series of multiple salivary tumors.

Of the seven patients with recurrent parotid mixed tumors, three patients had recurrences 15, 9 and 4 years from the time of simple enucleation of their tumors. Superficial parotidectomy in two patients and total parotidectomy in one patient were followed by no further recurrences for 5 years in two cases and for 3 years in one case. Two of these patients had their initial parotid surgery under local anesthesia in a doctor's office. Three patients had a second recurrence following simple excisions. The second recurrences came at a shorter interval from the second operations than did the first recurrences after the

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TABLE 1

Salivary tumors

Location	Number
Parotid gland	73
Submaxillary gland	7
Minor salivary gland	6
Sublingual	1
Total	87

TABLE 2

Types of parotid tumors studied

Tumor	Number
Mixed tumors Primary, 38 Recurrent, 7	45
Warthin's tumor	4
Hemangioma	1
Hemangiosarcoma	1
Carcinomas	22
Total	73

TABLE 3

Comparison of patients with mixed tumors and carcinoma of the parotid

	Age of Patients (Average)	Sex		Race		Side		Duration Symptoms (Average)
		M	F	W	N	R	L	
	yrs.							yrs.
Primary parotid mixed tumors (38)	39	17	21	29	9	24	14	2.7
Parotid carcinomas (22)	56	9	13	18	4	10	12	3

initial operations. These patients had total parotidectomies performed as their third operations, but these operations were performed too recently to allow adequate evaluation of the results.

The pathologic classification of the 22 parotid carcinomas is presented in table 4. Eleven of the patients, or 50 per cent, are dead. One patient is living with distant metastases from a parotid adenoid cystic carcinoma. Nine patients are living without evidence of recurrence from 2 to 6 years after their surgical treatment.

TABLE 4

Pathologic classification of parotid carcinomas

Types of Carcinoma	No. of Cases	No. of Deaths	Living 2 or more yrs.	Living with disease	Followed less than 2 yrs.
Mucoepidermoid	6	1	4	0	1
Adenocarcinoma	5	3	2	0	0
Undifferentiated	6	5	1	0	0
Acinic cell	2	0	2	0	0
Squamous cell	1	1	0	0	0
Adenoid cystic	1	0	0	1	0
Malignant mixed	1	1	0	0	0
Total	22	11	9	1	1

TABLE 5

Treatment of parotid carcinomas

Procedures	Number
Biopsy only	3
Parotidectomy with radical neck dissection	11
Parotidectomy	7
Parotidectomy with partial neck dissection	1
Total	22

In the treatment of the 22 patients with parotid carcinomas, three patients had only biopsies performed because their tumors were inoperable. Eleven patients had complete unilateral radical neck dissection with parotidectomy, and one patient had a partial radical neck dissection with parotidectomy. Seven patients had parotidectomies without neck dissections for low or moderate grade carcinomas without evidence of regional nodal metastases.

Irradiation was not employed in any case in these series except for recurrent or uncontrolled disease following surgery.

Six patients had mixed tumors of the submaxillary salivary gland. Four of these patients were seen primarily, and the two other patients had recurrent mixed tumors. One of the primary cases of a submaxillary mixed tumor was considered on frozen section to have a malignant

salivary tumor, and a radical neck dissection was performed.

One patient had a carcinoma of the submaxillary gland. This tumor was considered to be a benign mixed tumor at the time of its initial removal. After 3 years, the tumor recurred locally and was found, on review of the sections from the first operation, to be a mucoepidermoid carcinoma. Despite wide local removal and radical neck dissection at the second operation, the patient is now living with distant metastases.

These three recurrences of submaxillary salivary tumors, despite the technical ease of submaxillary surgery as compared with parotid surgery, illustrate the necessity for extreme care in surgical excision to prevent seeding the wound with salivary tumor cells.

Carcinomas of the sublingual gland are rare. Frazell[5] reported only one carcinoma of the sublingual gland in 870 tumors of the major salivary glands. A 57-year-old white woman was seen with a carcinoma of the sublingual gland which was treated by wide local removal and unilateral radical neck dissection. This patient has had no evidence of recurrence in 3 years.

In the minor salivary tumor group there was one female patient with a benign mixed tumor of the palate, and there were five patients with carcinomas originating in minor salivary tumors. Three of the latter patients were women and two were men. Two of these carcinomas arose from the lower alveolar ridge, one from the hard palate, one from the floor of the mouth invading the tongue posteriorly, and one from the buccal mucosa. Three were adenocarcinomas, two were mucoepidermoid carcinomas, and one was an adenoid cystic carcinoma. The case report of the latter patient is presented below.

CASE REPORT

A 42-year-old colored woman was seen in May, 1959, with a 6-month history of progressive difficulty in swallowing because of an enlarging mass elevating her tongue. Figure 1 illustrates the marked elevation of the tongue, extending almost to the hard palate. She had lost approximately 25 pounds in weight because of the difficulty she experienced in swallowing. A large, firm mass was felt elevating the entire tongue and presenting in the left submaxillary triangle. No significantly enlarged left or right cervical nodes were felt. Examination of the hypopharynx and indirect laryngoscopy were impossible because of the bulk of the tumor.

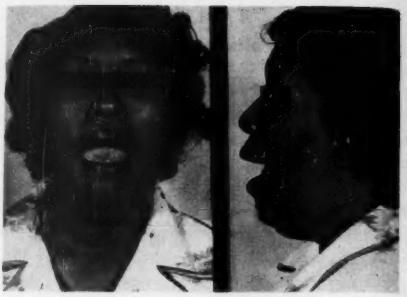

FIG. 1. Preoperative photograph showing marked elevation of tongue by tumor mass.

The patient had a microcytic hypochromic anemia compatible with an iron deficiency anemia. Her hematocrit was 24 volumes per cent and her hemoglobin was 7.1 gm. on admission to the hospital. She received 3000 ml. of blood transfusions in the 6 days preceding her operation.

Roentgen studies of her chest and of the left mandible were negative for metastatic disease or bone destruction.

The patient was operated upon on May 18, 1959. Biopsy of the mass presenting in the left submaxillary area revealed a salivary gland carcinoma, but the left submaxillary salivary gland was entirely normal in appearance. The tumor mass, which was grossly encapsulated except on its lingual aspect, measured 12 cm. in greatest diameter and extended to the base of the skull. A tracheostomy was performed because of the marked difficulty experienced in managing the airway as a result of the bulk of the tumor. A left radical neck dissection was performed. The posterior one-third of the tongue was invaded by the tumor and was resected with the tumor. The pharyngotomy opening was closed in layers after passing a Levin tube for feeding purposes.

Pathologic examination of the operative specimen was reported as showing a lobulated tumor mass 12 cm. in diameter with invasion of a zone of the posterior aspect of the tongue for 5.5 cm. at its base and 2.5 cm. in width. Microscopic examination revealed an adenoid cystic carcinoma (cylindroma) of salivary gland origin (fig. 2). A negative left submaxillary salivary gland and 16 negative lymph nodes were recovered from the left radical neck dissection.

The patient had marked drainage from the tumor site through the cervical wound for 3 weeks. The tracheostomy was removed after 2 weeks. The patient was able to discontinue tube feedings after 6 weeks, and she gained 35 pounds in the next 6 months (fig. 3). There has been no

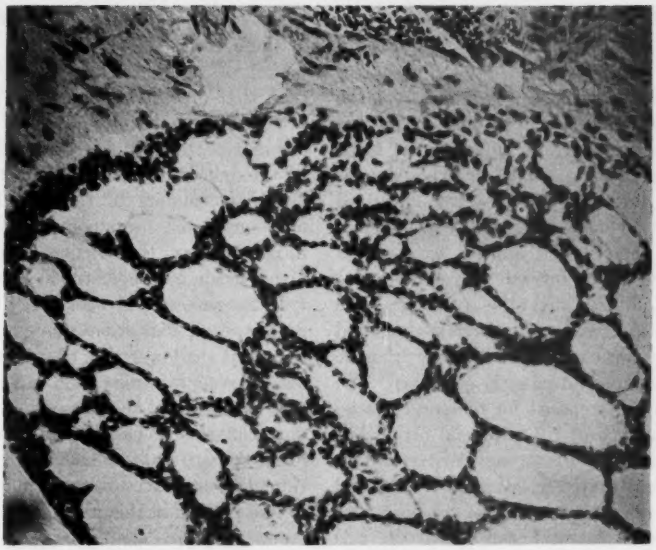

FIG. 2. Photomicrograph of adenoid cystic carcinoma ×100

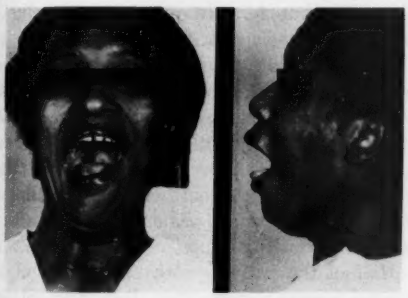

FIG. 3. Postoperative photograph

evidence of recurrence locally or remotely in the 16 months since surgical excision.

DISCUSSION

The minor salivary glands are distributed in the oral cavity and upper respiratory tract. Five of the six tumors in this small series arising from minor salivary glands were malignant.

The patient presented above has been extremely gratified to be able to eat and swallow again. The palliation obtained makes this type of procedure worthwhile even if subsequent recurrence does appear.

Fine[3] stated that among 98 recorded cylindromas or adenoid cystic carcinomas of minor salivary gland origin collected from the literature, 16 recurred locally 7 months to 5 years after therapy, and 17 metastasized. Late recurrences (more than 5 years postoperatively) and long survivals with recurrent tumor have been noted. McDonald and Havens[10] reported an average survival of 8½ years in 30 cases of cylindroma with recurrent tumor.

The four patients with papillary cystadenoma lymphomatosum or Warthin's tumors were all men over 40 years of age, and their tumors occurred in the lower part of their parotid glands. These benign neoplasms are said to comprise 2 to 6 per cent of all parotid tumors. These tumors may recur unless a wide zone of parotid tissue is removed with the tumor.

The high rate of recurrence of mixed tumors of the parotid has been related to incomplete excision of the tumor or to the spillage of tumor cells. Patey[12] showed that serial sections of the superficial lobe of the parotid after surgical removal revealed extension of foci of mixed tumor beyond the limits of apparent encapsulation in a significant number of cases.

Methods of surgical treatment of parotid tumors have evolved through the stages of enucleation to wider excision and finally to superficial or total parotidectomy. The recurrence rate in a large series of parotid mixed tumors following excision after a follow-up period up to

30 years was 32 per cent.[7] Many recurrences appear more than 3 years after the original surgery.

In addition to the complete removal of the tumor, the second major principle in parotid surgery is the avoidance of injury to the facial nerve.[9] The facial nerve should be exposed so as to protect it from injury in all cases of parotid surgery except for small, peripherally placed, well encapsulated tumors. The routine exposure of the main trunk of the seventh nerve anterior to the external auditory canal after it emerges from the stylomastoid foramen is the preferred method of exposure in our experience. A wide exposure through a Y-shaped incision is employed. The patient should be prepared for temporary facial paralysis after exposure of the facial nerve, but this weakness usually disappears in several days to 6 weeks postoperatively. Sacrifice of the facial nerve was performed only in the patients with highly malignant parotid carcinomas in this series.

Incisional biopsy was avoided to prevent the implantation of tumor cells. Local recurrences are frequent when tumor cells are spilled into the wound, and the recurrences are often in multiple foci.

Patients with recurrent mixed tumors of the salivary glands are much more difficult to treat than are patients who are seen primarily. In this group of 45 patients with mixed tumors of the parotid, seven patients had recurrent mixed tumors. Total parotidectomy is often required to control recurrent mixed tumors. Recurrence may occur more than 15 years after the initial surgery, so that no conclusions may be drawn about the recurrence rate in this recently treated series of cases. Beahrs *et al.*[1] have reported that none of 47 patients treated by them with conservative parotidectomy for primary mixed tumor and observed for 3 to 5 years has had a recurrence.

In this series, the average age of the patients with carcinoma of the parotid was 17 years older than the average age of the patients treated for primary mixed tumor. The duration of symptoms was not significantly different in these two series of cases. Thirty per cent of the parotid tumors were malignant.

Three of the 22 patients with carcinoma of the parotid had inoperable tumors when they were seen initially. Garcelon[6] reported that 25 per cent of the patients in his series with parotid carcinomas had inoperable tumors.

The extent of the surgical procedures employed in these cases varied, since the malignant potential of some parotid carcinomas is greater than that of others. Among the less malignant tumors of the parotid are the adenoid cystic carcinomas, the acinic cell carcinomas, and some of the mucoepidermoid carcinomas. The more malignant tumors of the parotid include the undifferentiated carcinomas, the malignant mixed tumors, the adenocarcinomas, and the higher grade mucoepidermoid carcinomas.

In the presence of cervical nodal enlargement, radical neck dissection was performed. Carcinomas of the lower part of the parotid in which extensive cervical dissection was required had an associated radical neck dissection performed. The more anaplastic carcinomas often metastasize distantly, so that radical neck dissection often cannot be employed in this group of patients.

One patient in this series, a 57-year-old white man, with an anaplastic parotid carcinoma survived 5 years after total parotidectomy with sacrifice of the facial nerve and radical neck dissection, but he died of metastases from a carcinoma of the splenic flexure of the colon.

A 27-year-old white man had a superficial parotidectomy for a low grade acinic cell carcinoma of the left parotid gland. Two years later a right superficial parotidectomy was performed for a tumor which proved to be a benign mixed tumor. This was the only example of bilateral salivary tumors which was encountered in these patients.

Half of the patients with carcinomas of the parotid reported above have died. The survival period is inadequate for any conclusions, but nine patients are living 2 to 6 years after operation without evidence of recurrent or metastatic parotid carcinoma.

In two cases, mixed tumors of the submaxillary gland were confused preoperatively with tumors of the tail of the parotid. In most series of cases of salivary tumors, there is a higher ratio of malignant to benign tumors in the submaxillary gland than in the parotid. In this small series, only one submaxillary carcinoma was encountered, whereas six patients with mixed tumors of the submaxillary gland were seen.

The two patients with recurrent mixed tumors of the submaxillary gland probably had spillage of tumor cells at the time of their initial operation. Difficulty in interpretation of the frozen section led to a radical neck dissection being performed

in one patient who subsequently proved to have a benign mixed tumor. Another patient was thought to have had a benign mixed tumor of the submaxillary gland removed. A recurrence 3 years later was found to be from mucoepidermoid carcinoma. Despite radical neck dissection and wide surgical removal, this patient was not cured by a second operation and is living with distant metastases.

SUMMARY

1. A total of 87 salivary tumors removed over a 5-year period in three hospitals in one community were reviewed.

2. Thirty per cent of the parotid tumors were malignant. The average age of the patients with carcinomas of the parotid was 17 years older than the average age of the patients with primary mixed tumors. The duration of symptoms was approximately the same in both series of cases.

3. One patient had bilateral nonsimultaneous parotid neoplasms. He had an acinic cell carcinoma of the left parotid, and 2 years later, he had a benign mixed tumor of the right parotid.

4. One patient had a rarely reported tumor, a carcinoma of the sublingual gland.

5. There were two recurrences of submaxillary mixed tumors which were thought to be due to seeding the wound with salivary tumor cells.

6. The case report of 42-year-old colored woman with a massive adenocystic carcinoma of the floor of the mouth invading the posterior one-third of the tongue was presented. The patient withstood the surgical treatment well and obtained much symptomatic relief in the short follow-up period of over 1 year.

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THE HORMONAL MEDIATION OF NEUROVASCULAR REFLEX ADJUSTMENTS: CATECHOL AMINE RESPONSE TO POSTURAL CHANGES IN MAN*

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GENERAL PHYSIOLOGY

Blood pressure is controlled by the interrelation of three factors: the output of the heart, the volume of circulating blood and the peripheral vascular resistance. Perhaps the most difficult of these to study is the peripheral vascular resistance. This resistance is produced mainly by the arterioles, but the capillaries and venules and even the larger arteries have been shown to play a part.[16] The amount of resistance depends on the degree of tone of these blood vessels, this being produced by the interrelation of at least three factors: the inherent properties of the vascular smooth muscle itself, the nervous influences coming to the blood vessel through the vasomotor nerves, and the effects of circulating hormones. By far the most important of these is the nervous system control through the vasomotor nerves to the blood vessels by vasoconstriction and, to some degree, vasodilation. Stimulation of the adrenergic nerves produces constriction of the peripheral blood vessels.

Although for many years it was believed that the impulses at the ends of postganglionic nerves were transmitted by the release of norepinephrine, just as vagal impulses were transmitted by the release of acetylcholine, not until relatively recently has the former been proved. It has now also been shown that norepinephrine is produced primarily by the sympathetic nerve endings, whereas epinephrine is produced primarily in the adrenal medulla.[10] This would further suggest that norepinephrine is the principal hormone regulating vasoconstriction, with epinephrine producing the greater effect as a circulating hormone by increasing the heart rate, vasodilation of certain abdominal vessels, and influence on the central nervous system. Norepinephrine appears to act locally, with epinephrine acting in a general way. Epinephrine is known to act directly on effector cells, producing stimulation in some and inhibition in others.

Normally, when a person undergoes marked postural change, as in rising from a recumbent to an erect position, the cardiovascular system must adjust promptly to compensate for the altered hydrostatic conditions. This is manifested by little change in the systolic blood pressure, an increase of 10 to 20 beats per minute in the heart rate, and an increase in the secretion of epinephrine and norepinephrine.[35] Failure to compensate for this increased hydrostatic pressure tends to cause blood vessels lying below the level of the heart to dilate with a shift in the distribution of blood to the dependent areas. Thompson *et al.*[36] showed an average loss of 11 per cent of the total plasma volume in a normal subject during prolonged standing. The maximal fluid loss occurred in from 20 to 30 minutes and was returned to the circulation in about the same time upon resumption of recumbency. The superficial veins are the ones shown to be more affected by the hydrostatic pressure, whereas there is little change in diameter of the deeper veins.[16] Evidence that postural hypotension is due to pooling of blood in the lower extremities is supported by the nullification of the hydrostatic effect by immersion of the subject in water.[15]

Postural Hypotension: The Clinical Entity

In 1925, Bradbury and Eggleston[4] first described the syndrome of postural hypotension as being characterized by a fall in blood pressure on assuming the erect position or on exertion; a slow, unchanging pulse; decreased sweating; diminished basal metabolism rate (BMR); intolerance to heat; increased reactivity to epinephrine; decreased reactivity to atropine; blood urea nitrogen (BUN) at the upper limits of normal; and no change in the electrocardiogram during the blood pressure fall. They con-

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cluded that the only adequate explanation for this fall in blood pressure was "paralysis of the sympathetic vasoconstrictor endings." Since that time, much subsequent work has been done to elucidate further the factors responsible for the syndrome of postural hypotension. Ellis and Haynes[7] also believed that postural hypotension was the result of the interruption of reflex sympathetic pathways because of its frequent association with neurologic disease. Stead and Ebert[34] studied postural hypotensive subjects and found that they had a normal blood volume and the same amount of peripheral pooling as normal controls. They concluded that the problem resulted from a failure of reflex control of arterial pressure to occur in the face of normal pooling, and not from excessive pooling.

In 1942, Springhurn and Hitzig[32] reported 50 carefully studied cases and concluded that postural hypotension was not a uniform clinical entity. The only constant feature that they noted was an immediate and persistent fall in blood pressure on standing. In two-thirds of the cases, the systolic blood pressure drop was 50 mm. Hg or greater. Vertigo and syncope were common, but in some patients the fluctuations in blood pressure correlated poorly with the symptoms and *vice versa*. In half the patients the pulse was slow and unchanging on assuming the erect position, and in the other half there was a normal to excessive acceleration of the pulse rate. Decreased sweating was present in only one-third of the cases. Seventy-five per cent of the cases were men and 50 per cent were 50 years or older. Only one-fourth of the cases had associated neurologic disease. In 75 per cent the condition presented in otherwise healthy individuals.

The most severe symptom of postural hypotension is syncope on standing. This is due to a lack of adequate cerebral perfusion pressure resulting in cerebral oligemia.[24] This form of syncope must be differentiated from the common faint or vasodepressor syncope. The latter can occur on tilting normal subjects upright to 60 degrees and is thought to be due to a vagotonic reaction superimposed on the normal sympathetic defense against orthostatic stress.[5] The signs and symptoms of cholinergic nerve activity preceding this type of syncope, together with peripheral vasodilation and the subsequent increase in limb flow,[5] are further evidence that this is a depressor response. The type of syncope in postural hypotension, by contrast, presents no signs and symptoms of excess cholinergic nerve activity and is due to a failure to compensate by reflex vasoconstriction for the pooling of blood in the dependent portions of the body.[4, 7, 17, 33, 34]

Relation of catechol amine elaboration to postural changes. It was noted above that the neurotransmitter of the postganglionic sympathetic nerve fibers is norepinephrine, and in recent years measurements of the epinephrine and norepinephrine elaboration have been correlated with the syndrome of postural hypotension. On tilting normal subjects to 75 degrees in the erect position, Sudin[35] noted a fourfold increase in the epinephrine and norepinephrine excretion in their urine. Later a higher "G" tolerance was correlated with an increased norepinephrine excretion in urine, as opposed to that in subjects with low urinary excretion of norepinephrine and a low "G" tolerance. No significant change was noted in the urinary epinephrine levels.[13]

Effect of adrenalectomy upon urinary excretion of epinephrine and norepinephrine in urine. Von Euler *et al.*[10, 11] found a marked decrease in urinary excretion of epinephrine following bilateral adrenalectomy, but no decrease in urinary norepinephrine excretion. This constituted further evidence that, whereas epinephrine is produced largely, if not entirely, in the adrenal medulla, norepinephrine is elaborated at the ends of postganglionic sympathetic nerve fibers everywhere.[39]

Effect of tilting on plasma epinephrine and norepinephrine levels in postural hypotension. Hickler *et al.*[17] performed tilt table studies on a series of postural hypotensive subjects and noted that they failed to increase their plasma level of norepinephrine and epinephrine during hypotensive episodes in the erect position. A control group maintained their blood pressure on tilting to 60 degrees in the erect position and had an increase in plasma norepinephrine and epinephrine levels. A subject in their series with a bilateral adrenalectomy failed to increase his plasma epinephrine level on tilting upright to 60 degrees, but did increase his norepinephrine level significantly. These findings would support the concept of a deficiency of norepinephrine as one of the factors which plays a role in the production of postural hypotension.

THE PRESENT STUDY

The purpose of the present study of 10 normal subjects and four with postural hypotension was to examine the effect of head up and head down tilting on the plasma levels of epinephrine and norepinephrine and to correlate these data with changes in blood pressure and pulse rate. It was anticipated that such investigation might further substantiate and extend our knowledge regarding the normal and abnormal physiology of the neuroendocrine role in the maintenance of vascular tone.

Procedure

The subjects included 10 healthy medical students and four patients known to suffer from postural hypotension. All were ambulatory and were studied 2 to 3 hours after either the noon or evening meal. First each was placed in the horizontal, supine position on a standard operating room table covered with a 1-inch thick sponge rubber mattress. The arms were extended on padded arm boards, and an inlying no. 18, 19 or 20 needle with stylet was placed in the antecubital vein of one arm, and a standard aneroid blood pressure cuff was placed on the opposite arm. Each subject remained in the above position for 30 minutes. The individual was then tilted head up to 60 degrees, and serial blood pressures and heart rates were recorded every 30 to 60 seconds. Venous blood samples were obtained just before tilting and at 2½, 5, 10, 20 and 30 minutes after tilting. The blood sample was drawn into a syringe containing 5 ml. of fluoride-thiosulfate solution and the plasma separated by centrifugation and decanted. The plasma was then stored at −15°C. until subsequent analyses of the epinephrine and norepinephrine concentration could be carried out by the Richardson modification[29] of the Weil-Malherbe and Bone method.[39] After head up tilting the subjects were again returned to the recumbent position and rested for 30 minutes. Following this, each was tilted head down to 60 degrees for 10 minutes. Again serial blood pressure and heart rates were recorded as before and blood samples obtained at 0, 2½, 5 and 10 minutes (table 2).

The only exception to the above procedure concerned subject E. G., who was tilted 10 degrees head up for 15 minutes and then 45 degrees head up for 15 minutes. She was tilted 60 degrees head down for 10 minutes. The reason for this change was because of the known severity of her symptoms.

Results

The data are presented in tables 1 and 2 and in figures 1 through 6.

Change in Vital Signs

Blood pressures, pulse rates and catechol amine levels vary considerably in normal subjects, and for this reason a total of 10 normal subjects was studied with a view to obtaining statistically valid results. The subjects ranged from 21 to 31 years of age and none had a history of syncopal spells. One woman and nine men were studied. All normal subjects selected were connected with the medical profession, since it was believed that their pulse rate and blood pressure value would be less affected by the novelty of the procedure than might be the case with persons to whom such procedures were entirely new.

The only normal subject to faint while being tilted was R. B., who fainted after 13 minutes of 60 degree head up tilting. Several other normal subjects (B. M., G. D., W. N. and S. A.) experienced episodes of increased sweating, nausea and vertigo. This occurred between 4 and 10 minutes and was not experienced after 10 minutes.

Of the abnormal subjects, J. F. was the only one to faint, and he did so after several minutes of head up tilting to 60 degrees. Subject M. J. experienced extreme nausea and vomiting after 28 minutes of 60 degree head up tilting. For this reason she was not tilted head down.

In table 1 are compared the control levels of blood pressure, heart rate, epinephrine and norepinephrine with the levels recorded at the time that the peak plasma norepinephrine level occurred post tilt. This peak level ranged from 2½ to 20 minutes in different subjects. These two times were used as reference points because of the importance of correlating the hemodynamic changes with the greatest change in plasma catechol amine levels, especially the level of norepinephrine. These values are recorded for both head up and head down tilting to 60 degrees.

Head up position. In the 10 normal subjects the systolic blood pressure showed an average fall of 7 mm. Hg (range, −22 to +15) between the two reference times. This corresponded to an

TABLE 1

Hemodynamics and catechol amine data on control subjects and postural hypotensive subjects, head up position

Subject	Age	Sex	History of Syncopal Spells	Blood Pressure					Heart Rate			Epinephrine				Norepinephrine				Response to Tilt
				Systolic			Diastolic													
				Control[a]	Post tilt[b]	Change	Control[a]	Post tilt[b]	Change	Control[c]	Post tilt[b]	Change	Control[c]	Maximal level post tilt	Change	Min. after tilt to time of level	Control[c]	Maximal level post tilt	Change	Min. after tilt to time of level	
	yrs.			*mm. Hg*			*mm. Hg*						*μg./L. plasma*			*min.*	*μg./L. plasma*			*min.*	
J. A.	24	M	0	122	110	−12	88	84	−4	60	76	+16	0	0	0		5.23	6.35	+1.12	5	
R. B.	24	M	0	140	120	−20	60	78	+18	86	112	+26	0.54	1.30	+0.76	0.33	0	2.60	+2.60	5	Fainted
T. M.	23	M	0	110	125	+15	62	78	+16	68	88	+20	2.7	0	−2.7		3.7	0	−3.7		
B. M.	23	F	0	114	112	−2	80	78	−2	66	80	+14	0	0.08	+0.08	5	0.64	2.6	+1.96	5	Nausea
C. S.	25	M	0	120	114	−6	50	82	+32	60	88	+28	1.07	1.69	+0.62	20	0	3.83	+3.83	5	
G. D.	31	M	0	116	110	−6	54	60	+6	68	84	+16	0.60	0.98	+0.38	2.5	0.80	1.0	+0.2	20	Nausea
G. S.	25	M	0	110	114	+4	60	70	+10	80	96	+16	0.98	1.00	+0.02	10	1.25	3.2	+1.25	20	
M. R.	21	M	0	114	102	−12	60	70	+10	76	80	+4	0.26	0.20	−0.06	2.5	0.46	1.50	+1.04	20	
W. N.	22	M	0	120	110	−10	64	60	−4	58	80	+22	0	0.52	+0.52	10	1.0	2.0	+1.0	10	Nausea
S. A.	24	M	0	112	90	−22	70	68	−2	72	80	+8	0	0.12	+0.12	20	0.16	1.91	+1.75	20	Nausea
E. G.[d]	48	F	+	106	76	−30	84	56	−28	84	88	+4	0.19	0.23	+0.04	20	0.89	0.92	+0.03	20	
J. F.[d]	18	M	+	122	55	−67	78	0	−78	64	32	−32	0.09	0.09			0	0.51	+0.51	10	Fainted
O. D.[d]	27	M	+	120	100	−20	90	70	−20	96	96	0	0	0			0.96	0.96	0	2.5	
M. J.[d]	33	F	+	118	70	−48	80	50	−30	78	94	+16	0.43	0.30	−0.13	2.5	0.20	0.38	+0.18	10	

[a] Level at time of drawing control blood sample.

[b] Level at time of drawing blood sample at peak norepinephrine response after tilt.

[c] Level just before tilt.

[d] Patients with postural hypotension.

TABLE 2

Hemodynamics and catechol amine data on control subjects and postural hypotensive subjects, head down position

Subject	Age	Sex	History of Syncopal Spells	Blood Pressure						Heart Rate			Epinephrine				Norepinephrine			
				Systolic			Diastolic													
				Control[a]	Post tilt[b]	Change	Control[a]	Post tilt[b]	Change	Control[a]	Post tilt[b]	Change	Control[c]	Minimal level post tilt	Change	Min. after tilt to time of level	Control[c]	Minimal level post tilt	Change	Min. after tilt to time of level
				mm. Hg			*mm. Hg*						*μg./L. plasma*[d]			*min.*	*μg./L. plasma*[d]			*min.*
J. A.	24	M	0	112	106	−6	88	82	−6	60	72	+12	0	0	0	5	5.93	0	−5.83	0
R. B.	24	M	0	116	124	+8	80	90	+10	84	98	+14	0.31	0	−0.31	5	0.64	0	−0.64	5
T. M.	23	M	0	122	118	+4	78	80	+2	64	68	+4	0.40	0.30	−0.10	2.5	2.20	0	−2.20	2.5
B. M.	23	F	0	88	120	+32	70	90	+20	72	84	+12	0.90	0	−0.90	5	0.70	0.38	−0.32	5
C. S.	25	M	0	110	130	+20	70	84	+14	64	60	+4	0.13	0.10	−0.03	5	0.77	0.31	−0.46	5
G. D.	31	M	0	110	104	−6	64	60	−4	80	72	−8	0.98	.54	−0.44	10	0.68	0	−0.68	10
G. S.	25	M	0	114	104	−10	70	68	−2	88	80	−8	0.86	0.80	−0.06	5	1.00	1.00	0	5
M. R.	21	M	0	110	106	−4	60	66	+6	76	80	+4	0.26	0	−0.26	5	0.46	0	−0.46	5
W. N.	22	M	0	118	98	−20	68	58	−10	60	64	+4	0	0	0	10	0.50	0.46	−0.04	10
S. A.	24	M	0	106	80	−26	60	60	0	64	76	+12	0	0	0	2.5	0.39	0.26	−0.13	2.5
E. G.[e]	48	F	+	110	142	+32	70	94	+24	88	88	0	0.08	0	−0.08	5	0.25	0	−0.25	5
J. F.[e]	18	M	+	120	138	+18	90	102	+12	84	60	−24	0	0	0	5	0.51	0.48	−0.03	5
M. J.[e]	33	F	+																	

[a] Level at time of drawing control blood samples.
[b] Level at time of drawing blood samples at minimal norepinephrine response after tilt.
[c] Just before tilt.
[d] Venous.
[e] Patients with postural hypotension.

average fall of 4 mm. Hg reported by Allen *et al.*[1] in 91 normal subjects. The four subjects with postural hypotension had an average fall of 41 mm. Hg (range, 30 to 67) between the two reference times. This corresponded to an average fall of 57 mm. Hg reported by Hickler *et al.*[17] on four subjects with postural hypotension.

The diastolic blood pressure of the 10 normal subjects showed an average rise of 8 mm. Hg (range, −2 to +32). This corresponded to an average rise of 5 mm. Hg in the large series by Allen *et al.*[1] The four postural hypotensive patients showed a fall in diastolic pressure with an average of 39 mm. Hg (range, 20 to 78) between the two reference times. This corresponded to an average fall of 13 mm. Hg in four patients with postural hypotension reported by Hickler *et al.*[17]

The heart rate rose in all the normal subjects, averaging 17 beats per minute (range, 5 to 28). This corresponded to an average rise of 15 reported by Allen *et al.*[1] The four subjects with postural hypotension had an insignificant fall, averaging 3 beats per minute. An essentially unchanged heart rate in association with marked hypotension has been observed before in postural hypotensive subjects.[7, 17]

Head down position. The reference time is changed now to compare the control levels with the levels recorded at the lowest level of norepinephrine recorded post-tilt. In the 10 normal subjects the systolic blood pressure showed an average fall of 2 mm. Hg (range, —26 to +32) between the two new reference times (table 2). One of the four subjects with postural hypotension was not tilted head down because of extreme nausea and vomiting. Of the three subjects tilted, an average rise of 21 mm. Hg (range 14 to 30) was recorded between the two reference times.

The diastolic blood pressure of 10 normal subjects showed an average rise of 3 mm. Hg (range, −10 to +20) between the two reference times. In the three postural hypotensive subjects an average rise of 15 mm. Hg (range, 8 to 24) was recorded.

The heart rate rose an average of 5 beats per minute (range, −8 to +14) in the normal subjects. No change between the two reference times was noted in two of the postural hypotensive subjects, but patient J. F. had a fall of 24 beats per minute.

Catechol Amine Values

Plasma catechol amine levels on tilting 60 degrees head up. The 10 normal control subjects had an average rise of 1.18 μg. per L. of plasma norepinephrine from 2½ to 20 minutes after tilting to 60 degrees head up (fig. 1). The average control level was 1.32 μg. per L., compared with a peak level averaging 2.50 μg. per L. Epinephrine levels presented a wandering base line and were essentially unchanged in the control group on tilting.

The four postural hypotensive patients had a characteristic hypotensive response, and the average rise in norepinephrine from 2½ to 20 minutes was 0.18 μg per L. (fig. 2). The average control level of plasma norepinephrine was 0.51 μg per L., compared with the peak level average of 0.69 μg. per L. The epinephrine level was essentially unchanged.

The normal group had, again, an average control plasma level of norepinephrine of 1.32 μg. per L., as contrasted with an average control level in the postural hypotensive group of 0.51 μg. per L. The average peak plasma level of norepinephrine in the normal group was 2.50 μg. per L., compared to 0.69 μg. per L. in the postural hypotensive subjects. The average rise in the normal group was a change of 1.18 μg. per L., as compared with a change of 0.18 μg. per L. in the postural hypotensive patients. These last two figures are the most significant, because they represent the average change in levels between the two reference points previously stated and not merely a comparison of absolute levels of norepinephrine.

Plasma catechol amine levels on tilting 60 degrees head down. The 10 normal subjects had an average fall in plasma norepinephrine level of 1.09 μg. per L. from 2½ to 20 minutes after tilting head down 60 degrees (fig. 3). This is in contrast to the three in the postural hypotensive group who had a fall of 0.09 μg. per L. in plasma norepinephrine (fig. 4). The average control level of plasma norepinephrine in the 10 normal subjects before tilting head down was 1.33 μg. per L., which correlated very favorably with the previous average control level of norepinephrine of 1.32 μg. per L. which was obtained in these normal subjects before tilting head up. This would indicate accuracy of the method used.

Ten normal subjects had an average of 0.21

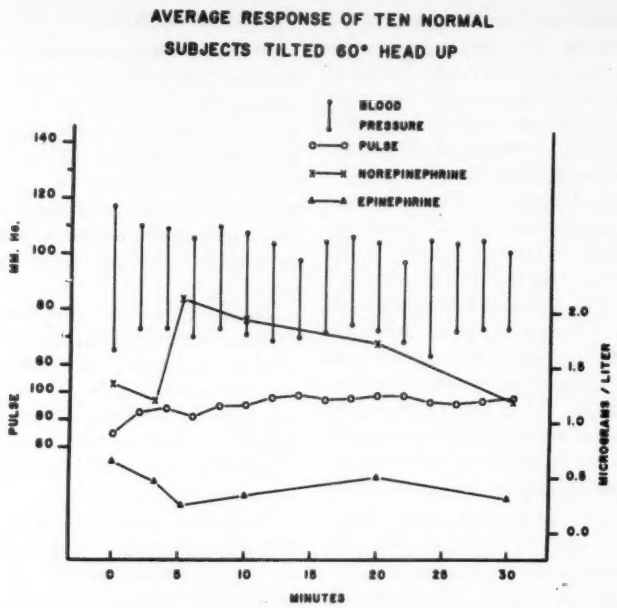

FIG. 1. The plasma norepinephrine level rose sharply within 5 minutes after head up tilting. The plasma epinephrine level declined slightly. The pulse rate increased, but the mean blood pressure remained essentially unchanged.

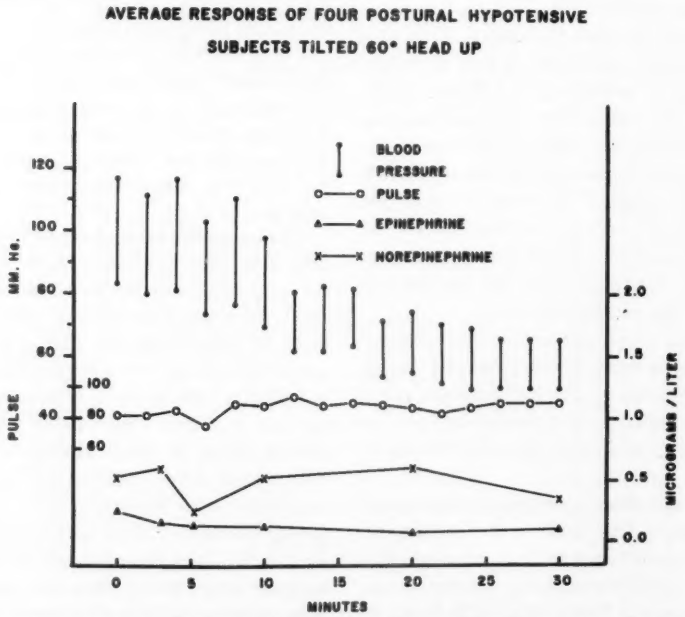

FIG. 2. Even though the blood pressure fell sharply when these patients with postural hypotension were tilted head up, the plasma norepinephrine level failed to rise as it did in normal subjects.

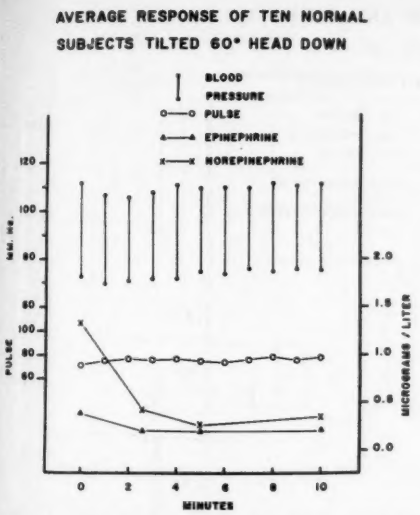

FIG. 3. The plasma norephinephrine level fell when these normal subjects were tilted head down. Blood pressure remained essentially unchanged.

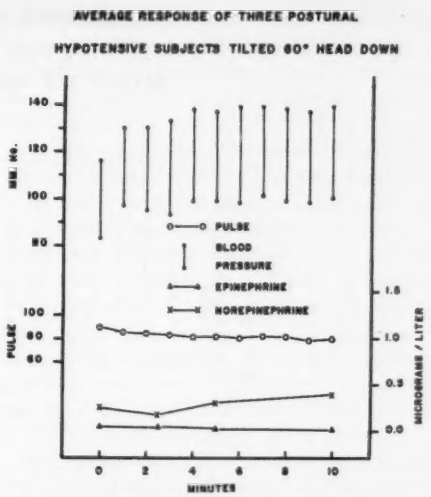

FIG. 4. The blood pressure rose when these patients with a diagnosis of postural hypotension were tilted head down. There was no change in the low plasma norepinephrine level.

μg. per L. fall in epinephrine, as contrasted with a 0.02 μg. per L. fall in epinephrine in three postural hypotensive subjects.

Statistical Evaluation

In nine of the 10 normal subjects the plasma norepinephrine level rose after 60 degrees tilting head up. The average rise was 1.18 μg. per L. of plasma. The mean control level as 1.32 μg. per L. $\pm$1.65, and the mean peak level was 2.50 μg. per L. $\pm$1.65. The difference of the means is significant (P <0.10). The four postural hypotensive subjects had an average plasma norepinephrine level rise of 0.18 μg. per L. on tilting 60 degrees head up. The mean control level was 0.51 μg. per L. $\pm$0.42, and the mean peak level was 0.69 μg. per L. $\pm$0.25. The difference of the means is not significant (P >0.45). The mean rise on tilting the 10 normal subjects 60 degrees head up was 1.18 μg. per L. $\pm$1.34, as compared with the mean rise of 0.18 μg per L. $\pm$0.20 in the four postural hypotensive patients. The difference between the mean rises in the plasma norepinephrine levels in each group is significant (P <0.02).

The 10 normal subjects had an average fall in their plasma norepinephrine level on tilting 60 degrees head down of 1.09 μg. per L. The mean control level of plasma norepinephrine was 1.33 μg. per L. $\pm$1.61, as compared with an average minimal post tilt level of 0.24 μg. per L. $\pm$0.30. There was a significant fall in plasma norepinephrine on tilting 60 degrees head down (P <0.04).

CASE REPORT

Patient E. G., a 48-year-old white woman, was first admitted to the University Hospital on February 23, 1958, because of weakness and fainting upon assuming the erect position and chronic diarrhea of 12 year duration. The syncopal attacks had begun insidiously about 7 years before admission and the first symptoms noted by the patient were morning dizziness and "splotches swimming before" her eyes. Further progression of these symptoms left the patient nearly completely incapacitated. She stated that she would have to crawl on her hands and knees to answer the doorbell and perform other such duties. A history of a hypersensitive reaction to epinephrine several years before was also elicited.

The diarrhea which began with frequent episodes was now constant, producing four to five loose, watery stools per day. Many physicians and clinics were consulted concerning this, but no cause could be determined. A cholecystectomy and exploratory laparotomy performed in another hospital several years before revealed no gastrointestinal tract abnormality. All laboratory and x-ray studies done at the University Hospital were essentially normal. The glucose tolerance curve was normal.

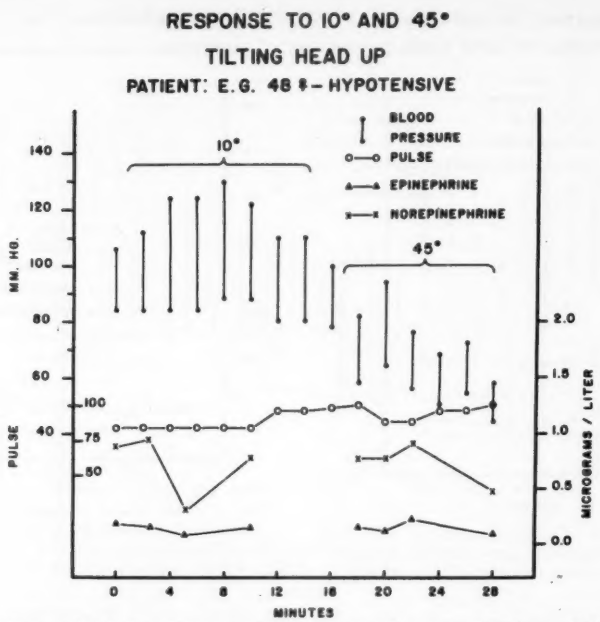

FIG. 5. This patient exhibited extreme postural hypotension upon head up tilting. There was no increase in the plasma norepinephrine level as there was in normal subjects.

Physical examination on admission revealed a well developed, thin white woman in no acute distress in the sitting position. The blood pressure was 100/70 mm. Hg in the supine position; 60/40 mm. Hg, sitting; and 40/0 mm. Hg, standing erect. The pulse rate was 80, 86 and 40 in the three positions, respectively. The rest of the examination showed the patient to be completely normal except for hyperactive deep tendon reflexes bilaterally.

Elastic bandages applied to the legs were of no benefit, but immersion in a tank of water up to the waist produced a normal blood pressure in the erect position. However, immediately after she emerged from the tank of water, the blood pressure dropped to 42/0 mm. Hg, and she fainted.

Treatment consisted of fitting the patient with a pressure garment.[30] By wearing this when erect she obtained marked improvement. She is now on a high protein, high salt (15 gm. per day) diet, and is taking 9α-fluorohydrocortisone, 1 mg. twice a day.

This case is presented to illustrate severe postural hypotension in which the patient was almost completely incapacitated before therapy and to reveal why she was studied somewhat differently from the other postural hypotensive patients.

Because of the severity of her symptoms, it was decided to study her at 10 degrees head up tilt to begin with and gradually to progress to the maximal tilt tolerated. It was found to our surprise that she had an elevation of blood pressure at 10 degrees (fig. 5). She was then tilted to 45 degrees over 3 minutes. Both systolic and diastolic pressure promptly and progressively fell. The study was stopped at 28 minutes when fainting seemed inevitable. With E. G., as with all the postural hypotensive patients, we were amazed at how asymptomatic she was with a blood pressure as low as 60/40 mm. Hg. Head down studies are shown in figure 6.

DISCUSSION

The plasma levels of epinephrine and norepinephrine were correlated with the hemodynamic response to postural changes in order to determine the part that these factors play in maintaining blood pressure in normal individuals, and the state of these factors in the syndrome of postural hypotension.

In the 10 normal subjects the blood pressure was maintained in correlation with a rise in the plasma norepinephrine level on tilting head up 60 degrees. The four postural hypotensive sub-

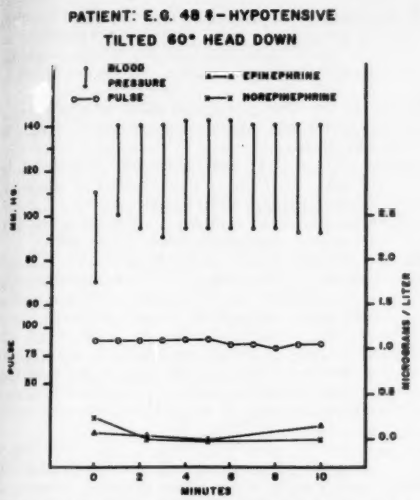

FIG. 6. This patient with postural hypotension could not make the circulatory adjustments necessary to prevent a rise in arm blood pressure on head down tilting. Contrast these findings with the blood pressure and norepinephrine responses to head down tilting in normal subjects (fig. 3).

jects had a characteristic fall in blood pressure and a slow, unchanging heart rate in correlation with a failure to increase the plasma level of norepinephrine. In addition, they demonstrated a much lower control level of norepinephrine than the 10 normal patients. The correlation of these data would indicate a lack of norepinephrine response in the syndrome of postural hypotension and a significant role of norepinephrine in the maintenance of an adequate blood pressure in the normal subjects.

In order to present a more inclusive view of the data used to obtain these results, a graphic illustration of the averages of the figures obtained in 10 normal subjects in the 60 degrees head up position is presented in figure 1. It depicts overall maintenance of the blood pressure, correlated with a rise in the plasma norepinephrine level. The rise of 8 mm. Hg diastolic pressure preceded the average peak level of norepinephrine by 3 minutes. This could indicate an immediate action of norepinephrine as it is released at the sympathetic postganglionic nerve endings, with the peak circulating blood level occurring later. This would further indicate that norepinephrine acts at the site of release, as opposed to the more widespread or general action that has been proposed for epinephrine. Also this would point to norepinephrine as a necessary, specific factor in the maintenance of normal hemodynamic response, as contrasted with epinephrine which serves as an emergency hormone and modifies other physiologic activities as well as circulatory activities.[14]

A composite graph of the postural hypotensive subjects' average response to tilting 60 degrees head up is presented in figure 2. It illustrates the characteristic fall in blood pressure and a slow, unchanging heart rate correlated with a failure of the norepinephrine level to rise. In addition, the control level of norepinephrine in the plasma is well below the control level found in normal subjects. The epinephrine levels in the postural hypotensive subjects are well below those of normal subjects, but these levels revealed no great change during the tilting of either group.

Both groups were then tilted 60 degrees head down to demonstrate the effect of removing the postural stress and allowing adequate cerebral perfusion.

In the 10 normal subjects the blood pressure remained essentially unchanged on tilting 60 degrees head down, and the plasma norepinephrine level decreased in all cases. This could imply that cerebral hypoxia may be one of the trigger mechanisms for release of norepinephrine. A graphic illustration of the findings on these 10 normal subjects is presented in figure 3. The three postural hypotensive subjects, by contrast, had an increase in blood pressure and very little change in their plasma levels of norepinephrine on tilting 60 degrees head down. This is graphically illustrated in figure 4.

The plasma norepinephrine levels were decreased in both groups when tilted head down and an adequate blood pressure was maintained, although the norepinephrine level of the postural hypotensives showed no significant change. This correlates with the finding of a decreased norepinephrine level when postural stress is removed. The postural hypotensive subjects had an increase in blood pressure and a very low control level of norepinephrine. They tended to be hypertensive in the head down position, possibly indicating their poor circulatory adjustment to any postural change.

SUMMARY AND CONCLUSIONS

1. Ten normal subjects showed a significant rise in plasma norepinephrine levels in response to tilting 60 degrees head up.

2. Plasma norepinephrine levels failed to in-

crease after head up tilting in four patients with postural hypotension.

3. The control plasma levels of norepinephrine in the postural hypotensive subjects were well below the control levels in the normal subject.

4. In normal subjects tilted 60 degrees head down, the plasma norepinephrine level fell promptly, demonstrating the effect of removing the postural stress and allowing adequate cerebral perfusion. This norepinephrine response was essentially absent in patients with postural hypotension.

5. The blood pressure level on tilting head down was essentially unchanged in normal subjects, but patients previously diagnosed as having postural hypotension exhibited an increase in arm blood pressure, indicating again their poor maintenance of normal vascular hemodynamics.

6. Further evidence is presented to strengthen the concept that epinephrine is produced largely by the adrenal medulla, but that norepinephrine is elaborated by the sympathetic postganglionic nerve endings everywhere.

7. The role of norepinephrine as an hormonal mediator in vascular response to postural stress is demonstrated.

Acknowledgments. We wish to express our appreciation to Dr. Herbert G. Langford for the use of his patients and for his helpful advice throughout this study. This work was supported by Army Contract no. DA-49-007-MD-627.

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Book Review

The editors of THE AMERICAN SURGEON *will at all times welcome new books in the field of surgery and will acknowledge their receipt in these pages. The editors do not, however, agree to review all books that have been submitted without solicitation.*

Fractures, Dislocations and Sprains. By PHILIP WILES, M.S., F.R.C.S., F.A.C.S., 67 pp., 519 illustrations, Little, Brown and Company, 1960.

This small book delivers an amazing amount of information on traumatic orthopedics. Its 67 pages are arranged according to anatomical region, presenting injuries of the upper limb, trunk and lower limb in that order. There is a preliminary section explaining the general principles of fracture healing and fracture treatment. Many excellent photographs and diagrams are used to simplify the discussion.

The wealth of material afforded by this work is amply illustrated by the section on knee injuries. A page describes by diagram and text eight types of fractures involving the tibial plateau and femoral condyles. The facing page illustrates these fractures by eight radiograph reproductions. Beneath the diagrams, there is a discussion of the fracture mechanism, treatment techniques and possible complications such as stiff knees, lateral popliteal palsy and osteoarthritis. The next page illustrates and describes ligamentous injuries of the knee in the same manner. A third page illustrates and discusses injuries of the patella and extensor mechanism. Each page of diagram and text faces a page of photographs.

In this manner, the important features of each type fracture are presented. The book deals with all the fractures one is likely to encounter, and will draw attention to peculiarities of the injury requiring special consideration. This is an excellent reference book to keep handy where injuries are seen. It will refresh the surgeon's memory of previous experience and guide his search for additional information.

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